# MAGNETIC RESONACE IMAGING KINEMATICS OF THE POSTERIOR CRUCIATE DEFICIENT KNEE

By

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*A thesis submitted in fulfilment of the requirements for the degree of Masters of Philosophy (Surgery)*

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# Declaration

I, Sivashankar Chandrasekaran, hereby declare that this submission is my own work and that it contains no material previously published or written by another person except where acknowledged in the text. Nor does it contain material that has been accepted for the award of another degree or diploma in any university.

In addition, ethical approval from the ACT Health Human Research Ethics Committee and the Australian National University Human Research Ethics Committee was granted for the studies presented in this thesis. Subjects were required to read a subject information document and informed consent was gained prior to data collection.

Signed, Sivashankar Chandrasekaran

Date 25th August 2013

As supervisor of Sivashankar Chandrasekaran's MPhil. work, I certify that I consider his thesis "Magnetic Resonance Imaging Kinematics of the Posterior Cruciate Deficient Knee" to be suitable for examination.

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## Abbreviations

| | |
|---|---|
| ACL | Anterior Cruciate Ligament |
| FFC | Flexion Facet Centre |
| MRI | Magnetic Resonance Imaging |
| PCL | Posterior Cruciate Ligament |
| PCL-d | Posterior Cruciate Ligament Deficient |
| PLC | Posterolateral Corner |
| TFC | Tibiofemoral Contact |

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# Thesis Abstract

It is important to understand the kinematics of the posterior cruciate ligament (PCL) deficient knee to aid in the operative and non-operative management of isolated PCL injuries and to correlate kinematic changes to the natural history of the injury. The majority of PCL injuries can be managed non-operatively with a quadriceps strengthening programme. However, not all rehabilitated patients are able to return to the same level of sports participation and a minority require operative intervention for symptomatic instability. Furthermore, arthroscopic and cadaveric studies demonstrate increased articular cartilage deformation in the medial compartment of the PCL deficient (PCL-d) knee. This project uses an in vivo MRI model to compare the kinematics of the PCL-d knee with knees of healthy controls and the contralateral uninjured knee. Analysis of the in vivo kinematics will provide information that will contribute to rehabilitation protocols and a better understanding of the pattern of articular cartilage deformation associated with the injury.

Cadaveric and static imaging studies demonstrate that isolated sectioning of the PCL increases posterior translation of the tibia particularly in the medial compartment of the knee. There have been no studies analysing the alteration of the tibiofemoral contact (TFC) point and flexion facet centre (FFC) in the PCL-d knee. The understanding of the kinematics of the TFC point and FFC is important as cartilage deformation occurs with alteration of tibiofemoral articulation. Furthermore, it is not known whether the contralateral knee of subjects with a PCL injury can be used as a valid control as anatomical variations have been shown to increase the risk of ligamentous knee injury.

The aim of this thesis is to compare the sagittal plane articulation of the tibiofemoral joint of the PCL-d knee with the knees of healthy controls and the contralateral uninjured knee. Difference in the articulation profile may help to explain the predominance of medial compartment degeneration seen arthroscopically in PCL injured knees. Analysis of the kinematics of the contralateral knee may help elicit associated kinematic abnormalities to PCL injuries and also test the validity of using the contralateral knee as a control.

The thesis consists of three papers that have been accepted for publication. Each paper has been presented as a chapter in the thesis. The first chapter is a review of the anatomical, biomechanical and kinematic findings of posterior cruciate ligament injury with respect to non-operative management. The chapter reviews the findings of cadaveric and in vivo imaging studies on the kinematics of PCL injury. The anatomy of the PCL is detailed and its primary and secondary restraining functions are reviewed. The function of the PCL is considered with that of the posterolateral corner structures as these injuries commonly occur together and most often result in long term disability. The findings of imaging studies on PCL deficiency are explored with reference to the mechanism of neuromuscular adaptation that allows the majority of athletes to return to sport after a period of non-operative rehabilitation. These experimental findings are compared to clinical findings in patients with PCL deficiency. Specific clinical parameters reviewed are gait analysis, muscular compensation, patterns of degenerative change, muscle strength and proprioception. The review finally draws together the findings of experimental studies on the

anatomical and kinematic effects of PCL injury and compares them to results of non operative rehabilitation outcomes and function.

The second chapter details a study to investigate sagittal plane articulation of the tibiofemoral joint of subjects with an isolated PCL injury. Magnetic resonance was used to generate sagittal images of ten knees of healthy controls and ten knees with isolated PCL injuries. The subjects performed a supine leg press against a 150N load. Images were generated at 15 degree intervals as the knee flexed from 0 to $90^0$. The TFC and the FFC were measured from the posterior tibial cortex. The TFC and FFC were significantly more anterior in the injured knee from 45 to $90^0$ of knee flexion in the medial compartment compared to the knees of healthy controls. The greatest difference between the mean TFC points of both groups occurred at 75 and $90^0$, the difference being 4 mm and 5 mm respectively. The greatest difference between the mean FFC of both groups occurred at $75^0$ of flexion, which was 3 mm. There was no significant difference in the TFC and FFC between the injured and knees of healthy controls in the lateral compartment. The study demonstrated that there is a significant difference in the medial compartment sagittal plane articulation of the tibiofemoral joint in subjects with an isolated PCL injury.

The third chapter compares the in vivo articulation of the knee of healthy controls to the contralateral knee of subjects with acute and chronic PCL injuries. Magnetic resonance was used to generate sagittal images of ten knees of healthy controls and ten knees with isolated PCL injuries (five acute and five chronic). The subjects performed a supine leg press against a 150N load. Images were generated at 15 degree intervals as the knee flexed from 0 to $90^0$. There was no significant difference

in the TFC and FFC between the knees of healthy controls and contralateral knee of subjects with acute and chronic PCL injuries in the medial and lateral compartments of the knee. The findings of this study suggest that in the setting of acute and chronic PCL injury the contralateral knee does not modify its articulation profile and the contralateral knee can be used as a valid control when evaluating the articulation of the PCL-d knee.

The thesis demonstrates that PCL injury is associated with altered sagittal articulation profile of the tibiofemoral joint. In particular the TFC and the FFC are located more anteriorly with knee flexion. These findings are in concordance with cadaveric studies and clinically may help explain increased incidence of medial compartment cartilage wear seen in arthroscopic studies of patients with PCL injuries. Furthermore, the contralateral knee can be used as a valid control for in vivo studies analysing the kinematics of PCL injury.

The main limitation of the study was the small sample size. However, despite this, altered kinematics of the PCL-d knee was still able to be demonstrated. Future work would include using the same model to analyse the effect of PCL reconstruction on restoring the kinematics of the injured knee.

# Introduction

The incidence of posterior cruciate ligament (PCL) injuries varies from 3% to 44% of all knee injuries[1-3]. Motor vehicle injuries and athletic injuries are the most common causes of PCL injuries[2]. The majority of athletes with an isolated PCL injury return to competitive sport with non-operative rehabilitation of the knee[4]. Physical therapy encompasses adequate knee stabilisation through compensatory muscle function to resist excessive posterior tibial translation[4-8]. Many studies suggest that with physiotherapy many athletes suffer little functional loss but some studies do report functional deterioration associated with time since injury[9-12]. In particular, arthroscopic studies demonstrate an increased incidence of medial and patellofemoral compartment articular cartilage degeneration in patients with PCL injuries[13-14]. An understanding of kinematics of the PCL injured knee will help in understanding the mechanics of articular deformation and the development of rehabilitation protocols to restore functional demands of the knee.

An understanding of the kinematics of PCL injury has been derived from cadaveric and in vivo imaging studies. Cadaveric studies have analysed the tensile strength[15], chondral deformation forces[16] and primary and secondary restraining functions of the PCL[17]. However, it is difficult to correlate the findings of cadaveric studies to clinical evaluation of the patient with a PCL injury because cadaveric studies cannot replicate the contribution to joint stability of surrounding neuromuscular structures. This issue is most accurately addressed with in vivo studies. These studies rely on imaging such as fluoroscopy, computer tomography and magnetic resonance to evaluate static and dynamic articulation profiles. These studies have explored translation and rotational displacement of the PCL injured knee and have demonstrated increased posterior translation of the medial tibia[18-19]. However, there have been no studies that have

analysed the articulation of the tibiofemoral joint with a PCL injury in terms of tibiofemoral contact (TFC) points. This is important as abnormal contact may lead to abnormal cartilage deformation forces.

An understanding of the kinematics of PCL deficiency is important for the diagnosis and management of patients with isolated PCL injury. The aim of this thesis is to explore the sagittal articulation profile of the injured and contralateral knee of patients with PCL deficiency. Specifically the aims of the thesis are:

1. To analyse the sagittal tibiofemoral joint articulation in terms of TFC and the position of the flexion facet centre (FFC) of patients with a PCL injury using magnetic resonance imaging (MRI).
2. To analyse sagittal plane tibiofemoral joint articulation in terms of TFC and the position of the FFC of the contralateral knee of subjects with PCL deficiency to determine whether it can act as a valid control or whether an abnormal articulation profile is associated with the contralateral knee.

The structure of the thesis consists of three chapters of individual journal publications. The first chapter is a literature review and the second and third chapter are studies that address each of the specific aims.

**Chapter One: A Review Of The Anatomical, Biomechanical And Kinematic Findings Of Posterior Cruciate Ligament Injury With Respect To Non-Operative Management.**

This chapter reviews the findings of cadaveric and in vivo imaging studies on the kinematics of PCL injury. The anatomy of the PCL is detailed and its primary and secondary restraining functions are reviewed. The function of the PCL is considered with that of the posterolateral corner structures as these injuries commonly occur together and most often result in long term disability. The findings of imaging studies on PCL deficiency are explored with reference to the mechanism of neuromuscular adaptation that allows the majority of athletes to return to sport after a period of non-operative rehabilitation. These experimental findings are compared to clinical findings in patients with PCL deficiency. Specific clinical parameters reviewed are gait analysis, muscular compensation, patterns of degenerative change, muscle strength and proprioception. The review finally draws together the findings of experimental studies on the anatomical and kinematic effects of PCL injury and compares them to results of non operative rehabilitation outcomes and function.

**Chapter Two: Magnetic Resonance Imaging Study Of Alteration Of Tibiofemoral Joint Articulation After Posterior Cruciate Ligament Injury.**

The aim of this study was to investigate sagittal plane articulation of the tibiofemoral joint of subjects with an isolated PCL injury. Magnetic resonance was used to generate sagittal images of ten knees of healthy controls and ten knees with isolated PCL injuries. The subjects performed a supine leg press against a 150N load. Images were generated at 15 degree intervals as the knee flexed from 0 to $90^0$. The TFC point and the FFC were measured from the posterior tibial cortex. The study showed that the contact pattern and FFC were significantly more anterior in the injured knee from 45 to $90^0$ of knee flexion in the medial compartment compared to the knees of healthy

controls. The greatest difference between the mean TFC points of both groups occurred at 75 and $90^0$, the difference being 4 mm and 5 mm respectively. The greatest difference between the mean FFC of both groups occurred at $75^0$ of flexion, which was 3 mm. There was no significant difference in the contact pattern and FFC between the injured and knees of healthy controls in the lateral compartment.

**Chapter Three: Sagittal Plane Articulation Of The Contralateral Knee Of Subjects With Posterior Cruciate Ligament Deficiency: An Observational Study.**

The aim of this study was to investigate whether the contralateral knee in a subject with a PCL injury can be used as a valid normal control because anatomical variations which may be present bilaterally, such as narrower intercondylar notch anatomy and variation in tibial slope have been identified as risk factors for anterior cruciate ligament injury. Furthermore, the contralateral knee may undergo adaptive changes as a result of the abnormal articulation pattern in the injured knee. Magnetic resonance was used to generate sagittal images of ten knees of healthy controls and ten knees with isolated PCL injuries (five acute and five chronic). The subjects performed a supine leg press against a 150N load. Images were generated at 15 degree intervals as the knee flexed from 0 to $90^0$. The TFC and FFC were measured from the posterior tibial cortex. There was no difference seen between the TFC and FFC between the knee of healthy subjects and contralateral knee of subjects with acute and chronic PCL injuries in the medial and lateral compartments of the knee. The findings of this study suggest there is no predisposing articulation abnormality associated with a PCL injury, in the setting of chronic injury the contralateral knee

does not modify its articulation profile and the contralateral knee can be used as a valid control when evaluating the articulation of the PCL-d knee.

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# Chapter One: A Review of the Anatomical, Biomechanical and Kinematic Findings of Posterior Cruciate Ligament Injury with Respect to Non-Operative Management

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Review

## A review of the anatomical, biomechanical and kinematic findings of posterior cruciate ligament injury with respect to non-operative management

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ABSTRACT

An understanding of the kinematics of posterior cruciate ligament (PCL) deficiency is important for the diagnosis and management of patients with isolated PCL injury. The kinematics of PCL injury has been analysed through cadaveric and in vivo imaging studies. Cadaveric studies have detailed the anatomy of the PCL. It consists of two functional bundles, anterolateral and posteromedial, which exhibit different tensioning patterns through the arc of knee flexion. Isolated sectioning of the PCL and its related structures in cadaveric specimens has defined its primary and secondary restraining functions. The PCL is the primary restraint to posterior tibia translation above 30° and is a secondary restraint below 30° of knee flexion. Furthermore, sectioning of the PCL produces increased chondral deformation forces in the medial compartment as the knee flexes. However, the drawback of cadaveric studies is that they can not replicate the contribution of surrounding neuromuscular structures to joint stability that occurs in the clinical setting. To address this, there have been in vivo studies that have examined the kinematics of the PCL deficient knee using imaging modalities whilst subjects perform dynamic manoeuvres. These studies demonstrate significant posterior subluxation of the medial tibia as the knee flexes. The results of these experimental studies are in line with clinical consequences of PCL deficiency. In particular, arthroscopic evaluation of subjects with isolated PCL injuries demonstrate an increased incidence of chondral lesions in the medial compartment. Yet despite the altered kinematics with PCL injury only a minority of patients require surgery for persistent instability and the majority of athletes are able to return to sport following a period of non-operative rehabilitation. Specifically, non-operative management centres on a programme of quadriceps strengthening and hamstring inhibition to minimise posterior tibial load. The mechanism behind the neuromuscular adaptation that allows the majority of athletes to return to sport has been investigated but not clearly elucidated. The purpose of this review paper is to draw together the findings of experimental studies on the anatomical and kinematic effects of PCL injury and summarise their relevance with respect to non-operative management and functional outcome in patients with isolated PCL deficiency.



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## Abstract

An understanding of the kinematics of posterior cruciate ligament (PCL) deficiency is important for the diagnosis and management of patients with isolated PCL injury. The kinematics of PCL injury has been analysed through cadaveric and in vivo imaging studies. Cadaveric studies have detailed the anatomy of the PCL. It consists of two functional bundles, anterolateral and posteromedial, which exhibit different tensioning patterns through the arc of knee flexion. Isolated sectioning of the PCL and its related structures in cadaveric specimens has defined its primary and secondary restraining functions. The PCL is the primary restraint to posterior tibia translation greater than $30^0$ and is a secondary restraint less than $30^0$ of knee flexion. Furthermore, sectioning of the PCL produces increased chondral deformation forces in the medial compartment as the knee flexes. However, the drawback of cadaveric studies are that they can not replicate the contribution of surrounding neuromuscular structures to joint stability that occurs in the clinical setting. To address this, there have been in vivo studies that have examined the kinematics of the PCL deficient knee using imaging modalities whilst subjects perform dynamic manoeuvres. These studies demonstrate significant posterior subluxation of the medial tibia as the knee flexes. The results of these experimental studies are in line with clinical consequences of PCL deficiency. In particular, arthroscopic evaluation of subjects with isolated PCL injuries demonstrate an increased incidence of chondral lesions in the medial compartment. Yet despite the altered kinematics with PCL injury, only a minority of patients require surgery for persistent instability and the majority of athletes are able to return to sport following a period of non-operative rehabilitation.

Specifically, non-operative management centres on a programme of quadriceps strengthening and hamstring inhibition to minimise posterior tibial load. The mechanism behind the neuromuscular adaptation that allows the majority of athletes to return to sport has been investigated but not clearly elucidated. The purpose of this review paper is to draw together the findings of experimental studies on the anatomical and kinematic effects of PCL injury and summarise their relevance with respect to non-operative management and functional outcome in patients with isolated PCL deficiency.

## Introduction

The posterior cruciate ligament (PCL) is twice as strong as the anterior cruciate ligament[1] and is the primary restraint to posterior tibial translation[2] but management of PCL injuries has been less extensively studied than its anterior counterpart[3]. Cadaveric studies have detailed the anatomy of the PCL. It consists of two components, the anterolateral and posteromedial bundle which demonstrate different strain rates at different degrees of knee flexion[4]. Cadaveric studies have also analysed the tensile strength[5], chondral deformation forces[6] and primary and secondary restraining functions of the PCL[7]. However, it is difficult to correlate the findings of cadaveric studies to clinical evaluation of the patient with a PCL injury because cadaveric studies cannot replicate the contribution to joint stability of surrounding neuromuscular structures. This issue is most accurately addressed with in vivo studies analysing the kinematic profile of the knee in patients with PCL injuries. These studies rely on imaging such as fluoroscopy, computer tomography and magnetic resonance to evaluate static and dynamic articulation profiles.

The incidence of PCL injuries varies from 3% to 44% of all knee injuries[8-10]. Motor vehicle injuries and athletic injuries are the most common causes of PCL injuries[9]. Athletic injuries are most likely to result in an isolated PCL injury whereas motor vehicle accidents produce multiple-ligament damage[11]. The majority of athletes with an isolated PCL injury return to competitive sport with non-operative rehabilitation of the knee[12]. Physical therapy encompasses adequate knee stabilisation through compensatory muscle function to resist excessive posterior tibial translation[12-16]. Common programmes involve a 2 to 4 week period of immobilisation with the knee

braced in full extension. Extension reduces the tibiofemoral joint by preventing posterior sag and diminishes the effects of gravity and hamstring muscle contraction on tibial translation. Following this, quadriceps muscle strengthening exercises are encouraged and the use of hamstring muscles is prohibited to minimise posterior tibial load. Many athletes then return to sport one to three months after injury. Athletes are advised to return to sport once they have regained their full range of motion and 90% of the strength of the contralateral knee[8]. Many studies suggest that with physiotherapy many athletes suffer little functional loss but some studies do report functional deterioration associated with time since injury[17-20].

The purpose of this review paper is to provide an overview of the anatomy, biomechanics and kinematics of the PCL injured knee and discuss the basic science findings with respect to the results of non-operative treatment and the functional outcome of isolated PCL injuries.

## Anatomy

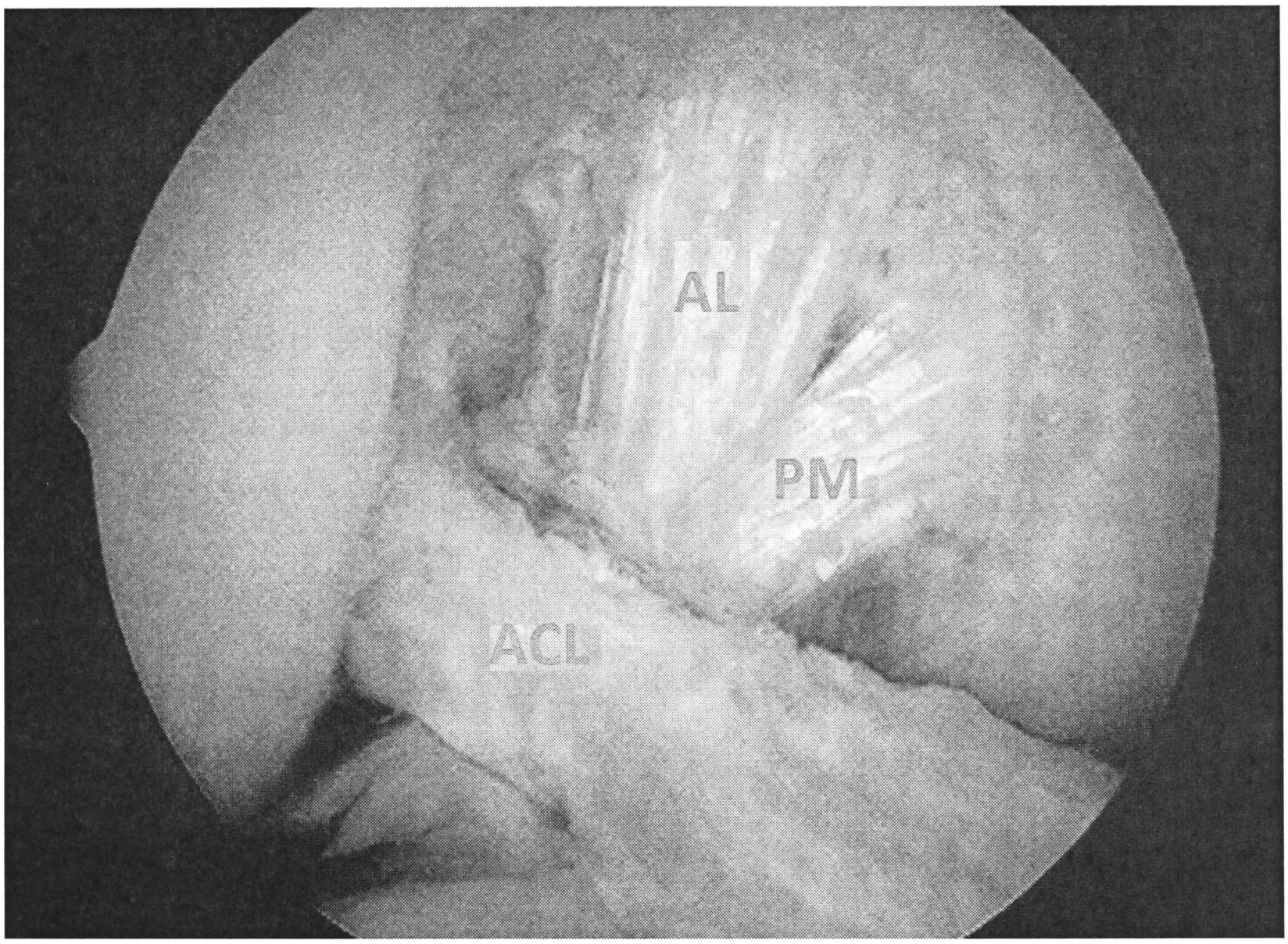


Figure 1-1: PCL anatomy showing the relationship of the posteromedial (PM) and anterolateral (AL) bundles to the anterior cruciate ligament.

The PCL arises from the posterior tibia 10 mm distal to the joint line and extends in an anteromedial direction to insert on the lateral surface of the medial femoral condyle[22-23]. The PCL is between 32 and 38 mm long[1]. It has a cross sectional area of 48 $mm^2$ at its midsubstance level[24]. The tibia and femoral insertion sites of the PCL are approximately three times larger than its midsubstance cross sectional area. The PCL arises from the tibia within a depression between the posterior aspect of the medial and lateral plateaus, approximately 1cm distal to the joint line, at an average width of 13 mm[25]. Its femoral attachment is semicircular and horizontal in direction,

with a footprint of 128 $mm^2$. It terminates 3 mm proximal to the articular cartilage margin of the femoral condyle[23]. Cadaveric studies have demonstrated that the PCL consists of two functional components[3]. These are named as the anterolateral and posteromedial bundles with reference to their attachments from the tibia to the femur (Figure 1-1). The footprint of these two bundles on the tibia and femur is approximately equal in size. The cross-sectional area of the anterolateral bundle is twice as large as the posteromedial bundle. The bundles have different tensioning patterns through the arc of knee flexion[4]. The anterolateral bundle is lax in extension and progressively becomes more taught as the knee flexes. In contrast, the posteromedial bundle is lax in flexion and becomes more taught as the knee extends. Although the two bundle functional division of the PCL is most commonly accepted, there also exists alternative anatomical descriptions of the PCL including three- and four- bundle divisions as well as a continuum of PCL fibre orientation[26]. However, further studies are required to investigate the validity of these alternate anatomical descriptions.

Two meniscofemoral ligaments are anatomically related to the PCL. These ligaments originate from the posterior horn of the lateral meniscus, bifurcate around the PCL, and insert anterior and posterior to the PCL on the medial femoral condyle[27]. The anterior of the two ligaments is named the ligament of Humphrey and the posterior of the two ligaments is named the ligament of Wrisberg. The anatomical description of these two ligaments in the literature is highly variable with discrepancies regarding their prevalence[27-28]. The biomechanical function of the meniscofemoral ligaments have not been fully characterised, but it has been suggested that they contribute to the

anterior-posterior rotatory stability of the knee and provide stability to the lateral meniscus[5].

Branches of the middle geniculate artery provide the major blood supply to the PCL[23]. The PCL has a relatively avascular portion in the middle third of the ligament, suggesting that the proximal and distal portions of the PCL have more potential for healing than midsubstance tears[29].

Clinical evaluation of posterolateral corner structures is important because they are damaged in 60% of PCL injuries[30]. The posterior lateral corner (PLC) can be divided into static and dynamic stabilisers that produce posterolateral stability to the knee. The static stabilisers include the lateral collateral ligament (LCL), the fabellofibular ligament, the posterior horn of the lateral meniscus, the lateral part of the posterior capsule and the PCL[31-32]. The dynamic stabilisers of the PLC include the popliteus complex, the biceps tendon and the iliotibial tract[33]. Of the PLC, the LCL and popliteus complex are the primary restraints to varus and external rotation of the knee, respectively[31-32]. The popliteus complex consists of the popliteofibular ligament, the popliteotibial fascicles, and the popliteomeniscal fascicles, all of which arise from the popliteus tendon and insert onto the fibula, the tibia, and the meniscus, respectively[33]. The popliteus muscle tendon unit arises from the posteromedial surface of the proximal tibia and inserts in the popliteal groove of the lateral surface of the lateral femoral condyle[33]. The popliteofibular ligament is the primary restraint to posterolateral instability or external rotation and recurvatum[31-32]. The main biomechanical function of the muscle tendon unit is to internally rotate the tibia[31-32].

## Cadaveric studies

The biomechanical properties of the PCL have largely been derived through the use of in vitro cadaveric models. In these models, cadaveric knees are tested before and after sectioning of the PCL using either simulated muscle loads or anterior, posterior and rotational loading[7]. Sectioning of the PLC, meniscofemoral ligaments or medial compartment structures further helps to determine the primary and secondary restraining functions of the PCL[34]. The main drawback of in vitro cadaveric studies is that they can not exactly replicate the neuromuscular contribution to joint stability that occurs in the clinical setting of PCL injury.

In vitro studies have demonstrated that isolated sectioning of the PCL significantly increases posterior tibial translation[35-38]. The studies have shown that the PCL is the primary restraint to posterior tibial translation at $90^0$ of knee flexion with the amount of translation increasing from 3.8 to 14.1 mm[35, 38]. It is not entirely clear as to when isolated sectioning of the PCL increases posterior translation. One study reported no posterior translation under $25^0$ flexion[36], two reported posterior tibial translation only above $60^0$ flexion[35, 37], while others report increased translation for angles ranging between 0 and $120^0$ flexion[38].

There are conflicting results from cadaveric studies on PCL function in restraining tibial rotation. Five studies have reported that isolated PCL sectioning does not increase tibial external rotation[35-36, 39-41], while four demonstrated significant increases ranging from 2.7 to $4^0$ [7, 34, 42-43]. One study reported that isolated PCL sectioning increased tibial internal rotation at $90^0$ of flexion[44].

To help understand the biomechanics of combined ligament injuries there have been several studies that have analysed the effects of sectioning other ligaments in PCL deficient models. Sectioning of the meniscofemoral ligaments increased posterior translation of the tibia but not anterior translation or rotation[34]. Specifically, meniscofemoral ligaments contribute 28% of the resistance to posterior tibial draw at $90^0$ of flexion in the intact knee and 70% in the PCL deficient knee[34]. Combined sectioning of PLC in the PCL deficient knee increased varus instability from 0.9 ± $1.0^0$ to 27.5 ± $6.4^0$ at $70^0$ flexion and internal/external rotational laxity from 30.9 ± $6.5^0$ to 41.7 ± $4.9^0$ [41]. In specimens with a cut PCL and lateral collateral ligament, sectioning of the popliteus tendon and posterolateral capsule increased posterior translation of the tibia to 20.4 mm at $90^0$ of knee flexion[40].

In vitro studies have also demonstrated increased joint contact pressure in the medial compartment of the PCL deficient knee. One study reported a significant increase at 60 and $90^0$ of flexion but no changes at 0 and $30^0$ of flexion[6]. Another study reported a mean increase of 52% of medial compartment pressure regardless of flexion angle[45]. This finding correlates with increased incidence of articular cartilage degeneration in the medial compartment of subjects with isolated PCL injury[46-50].

## In vivo studies

The advantage of clinical studies over cadaveric studies is that clinical studies take into consideration the contribution of neuromuscular structures when analysing isolated ligament deficiency. Clinical studies rely on using various imaging

modalities to analyse knee kinematics. Sagittal plane articulation of the tibiofemoral joint can be described by tibiofemoral contact mapping and the position of the flexion facet centre in normal[51], injured[52] or osteoarthritic knees[53-54]. The flexion facet centre is defined as the centre of the posterior circular surface of each femoral condyle in the sagittal plane. Its position is identified relative to the posterior tibial cortex.

Review of the kinematics literature indicates that PCL injury increases posterior translation of the tibia relative to the femur during flexion[2, 52]. The extent of posterior translation varies among the studies and may reflect different joint loading protocols. Castle *et al.* used fluoroscopy to assess sagittal plane translation in subjects with a PCL deficient knee performing a static squat at different degrees of knee flexion[2]. They reported that between 70 and $90^0$ of knee flexion the mean increase in posterior translation of the PCL deficient knee compared to the uninjured knee was 7.4 mm. At low flexion squats (32-$50^0$ of flexion) the increase in posterior tibial translation was not as marked being only 2.1 mm. Logan *et al.* analysed sagittal tibiofemoral articulation on MRI in subjects with PCL deficiency performing a weight bearing squat and undergoing a posterior drawer examination[52]. They used the flexion facet centre rather than the tibiofemoral contact to analyse the sagittal plane motion of the knee. They reported significant posterior subluxation of the medial tibia throughout the flexion arc from 0 to $90^0$ and 10.1 mm of posterior displacement with the posterior draw at $90^0$ of flexion.

No clinical studies have demonstrated significant varus-valgus instability with PCL deficiency. In particularly, gait and stepping analysis has demonstrated no significant difference between PCL deficient subjects and normal limbs in varus-valgus

instability or in internal/external rotation[55-57]. This is in contrast to some cadaveric studies. This may be explained by the way in which posterior soft tissues become compressed as the knee flexes greater than $90^0$ in in vivo studies whereas in in vitro studies these additional structures are often removed accounting for the greater rotational laxity.

## Concomitant posterolateral corner injuries

The PLC is the primary restraint against external rotation, with the secondary restraint being the PCL. Non-operative management has limited place in concomitant PCL/PLC injuries. Loss of the PLC also further increases the degree of posterior laxity. Some authors believe that grade 3 PCL tears inherently involve adjacent structures, most commonly the PLC[14, 58]. PCL tears are graded based on posterior subluxation of the medial tibial plateau relative to the medial femoral condyle with the knee in $90^0$ of flexion[58]. A grade 1 tear is 1-5mm of posterior translation of the tibia, a grade 2 tear is 6-10mm of posterior tibial translation and a grade 3 tear is greater than 10mm of posterior tibial translation. Patients are less likely to adapt and are more likely to have an unstable joint with concomitant PCL/PLC injuries. Although there is a paucity of literature regarding non-operative management of concomitant PCL/PLC injuries, recent review articles recommend early surgical repair in such settings.[59-60]

## The impact of altered kinematics of PCL injury on long term outcomes and degenerative change

Table 1-1:Gait-Analysis Studies

| Outcome Measure | Principal Author | LOE | Significant Results |
|---|---|---|---|
| Gait and Stepping Analysis - Stair Ascent | Hooper[56] | III | No significant difference. |
| | Jonsson[57] | III | No significant difference. No change in extension, abduction-adduction, during step-up test between injured and uninjured knees. |
| Gait and Stepping Analysis - Walking | Fontbote[55] | II | PCL-d (posterior cruciate ligament deficient) knees (0.78N) and uninjured knees (0.78N) had significantly greater vertical ground reaction force at midstance gait compared with healthy controls (0.70N). |
| | Hooper[56] | III | PCL-d knees (16°) exhibited a decreased mid-stance excursion angle when compared with controls (18°). PCL-d knees (0.57) exhibited greater peak extensor torque when compared with |

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| | | | controls (0.50). |
| Gait and Stepping Analysis - Stair Descent | Hooper[56] | III | PCL-d knees (4°) averaged lower knee flexion at toe contact while descending stairs when compared with controls (6°). |
| Gait and Stepping Analysis - Terminal stance | Tibone[61] | III | PCL-d knees had a significant 10% lower vertical force in the terminal stance during free walking compared with uninjured knees. |
| Gait and Stepping Analysis - Vertical Ground Force | Fontbote[55] | II | PCL-d knees (60.5 body weight/s) and uninjured knees (58.2 body weight/s) had significantly lower vertical ground reaction force loading rates compared with healthy controls (94.4 body weight/s). |

LOE: Level of Evidence

Various gait-analysis studies show no significant difference in kinematics and kinetics between PCL injured, contralateral uninjured and healthy control knees with respect to self-paced walking, running, vertical drops, stair ascent and step ups[55-57] (Table 1-1). Outside of preferred gait speed however, PCL-d knees flex less, show lateral thrust with less valgus moment. In the midstance for fast walking there was significantly decreased excursion angle in the PCL-d knee compared to the contralateral side ($16^0$ vs. $18^0$) and during stair descent, knee flexion at toe contact was significantly less in PCL-d subjects compared to healthy controls ($4^0$ vs. $6^0$)[56]. PCL-d subjects demonstrated decreased load rate compared to healthy controls when engaging in vertical drop tasks and greater vertical ground reaction force at midstance

gait[55]. Tibone *et al.* reported that during the terminal stance for free walking the vertical force and fore-aft shear force are significantly lower in the PCL-d limb compared to the contralateral side[61]. Maximum stress on the PCL occurs during terminal stance, these findings suggest that patients have modified their gait to decrease forces across a PCL-d knee. Whilst there is little difference in kinematics for self-paced activities, more physically demanding activities highlight more significant compensatory differences (Table 1-1).

Table 1-2: Muscular Compensation

| **Outcome Measure** | **Principal Author** | **LOE** | **Significant Results** |
|---|---|---|---|
| Muscular Compensation | Cain[62] | IV | Early contraction of the quadriceps in the PCL-d knee (24.9%) relative to the uninjured knee as recorded by EMG studies. |
| | Inoue[63] | III | Early contraction of the gastrocnemius muscle at both 30Hz (75ms vs. 42ms) and 60Hz (72ms vs. 41ms). |
| | Tibone[61] | III | Early contraction of the gastrocnemius-soleus complex during stance phase. |

LOE: Level of Evidence

Literature suggests that such compensatory muscle activity occurs in PCL-d subjects and involves both quadriceps and gastrocnemius activation with the objective being hamstring avoidance (Table1-2). Cain and Schwab reported on the EMG findings of

a footballer with an isolated PCL deficiency[62]. They demonstrated that the quadriceps in the injured leg contracted 20% earlier in the gait cycle than the uninjured leg during jogging. Tibone *et al.* reported early activation of the gastrocnemius-soleus complex during the stance phase of gait in 20 PCL subjects, 10 with reconstructions and 10 non-operatively treated[61]. Early onsets occurred for the reconstructed group in 50% of subjects for both fast and free walking whereas in the non-operative group it occurred in 38% of subjects for fast walking and 75% for free walking. Inoue *et al.* analysed 12 subjects with isolated PCL injuries and recorded EMG and torque during isokinetic contractions[63]. Through a flexion arc of 0 to $90^0$, contractions were performed at 30 and 60 Hz. They demonstrated a significant earlier activation of the gastrocnemius in the PCL injured knee during generation of flexion torque at both isokinetic velocities (Table1-2).

Table 1-3: Degenerative Changes

| **Outcome Measure** | **Principle Author** | **LOE** | **Significant Results** |
|---|---|---|---|
| Degenerative Changes - Articular | Boynton[46] | III | Increasing time intervals from original injury significantly correlated with degenerative changes on plain radiograph. At a mean follow-up period of 13.2 years, 60% of patients had degenerative arthritic changes on plain radiograph in the PCL-d knee. No statistical significant correlation between degree of laxity and knee score |

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| | | or radiographic evidence of degenerative arthritic changes. |
| Keller[47] | IV | Increasing time intervals from original injury significantly correlated with degenerative changes on plain radiograph. At a mean follow-up period of 6 years, <2 years post-injury, 100% had normal/grade 1 changes, whilst at >15 year follow-up, >80% developed grade II/III changes. |
| Parolie[48] | IV | No statistical relationship between increasing time intervals from original injury and severity of degenerative changes on plain radiograph. At a mean follow-up period of 8.4 years, 64% of patients had no radiographic evidence of arthritic disease. |
| Shelbourne[49] | II | At a mean follow-up period of 5.4 years, 15% of patients had degenerative arthritic changes on plain radiograph in the PCL-d knee, not statistically significantly greater compared with 5% of patients who had degenerative changes in the uninjured knee alone. Degenerative changes occurred most |

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| | | | commonly in the medial compartment. |
| | Strobel[64] | IV | Increasing time intervals from original injury significantly correlated with greater$^{0}$ of degenerative cartilage change as determined by arthroscopy, with an overall incidence of 67.4%. <1 year post-injury, 60.9% had normal/grade 1 changes, whilst injuries of >5 years duration, >55.5% developed grade II/III changes. |
| Degenerative Changes - Compartment | Boynton[46] | III | Degenerative changes occurred most commonly in the medial compartment (53%), and then the lateral tibiofemoral compartment. |
| | Keller[47] | IV | Degenerative changes appeared to occur in the medial compartment, involving lateral and patellofemoral compartments over time. |
| | Parolie[48] | IV | Degenerative changes appeared to occur in the medial compartment. |
| | Strobel[64] | IV | Degenerative changes occurred most commonly in the medial and patellofemoral compartments. |
| Degenerative Changes - | Hamada[65] | IV | 28% of cases had associated meniscal tears. Most common were lateral |

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| Meniscal | | | meniscal tears in 18% of patients. |
| | Keller[47] | IV | 5% of cases subsequently developed meniscal tears. |
| | Shelbourne[49] | II | 2% of cases subsequently developed meniscal tears. |

LOE: Level of Evidence

PCL injuries are known to be associated with a variety of pathologies including meniscal and chondral degeneration at the time of injury[46] (Table 1-3). Hamada arthroscopically evaluated acute isolated PCL injuries in 61 patients and exhibited concurrent articular cartilage damage and meniscal tears in 52% and 28% of patients respectively[65]. Five studies report on the subsequent development of degenerative changes as a result of non-operative management. Studies have demonstrated that the prevalence of articular degeneration ranges from 15%[49] to 88%[47] and increases from the time of injury. Shelbourne *et al.* conducted a prospective trial of non-operatively treated isolated PCL injuries, and found that 15% had developed arthritic changes on plain radiograph in only the PCL-d knee. This was not significantly different to degenerative changes in control knees. However, various other studies utilising plain radiographs and arthroscopy have reported significant articular degenerative changes in PCL-d knees[46-47, 64]. The difference between results may be explained by a combination of factors including: inclusion of patients who had subsequent surgical intervention in the follow-up period and a lengthier follow-up period. Shelbourne *et al.* reported a mean follow-up period of only 5.4 years compared with Boynton *et al.* who reported a mean period of 13.2 years (Table 1-3).

With regards to the location of the cartilage defects, Strobel *et al.* reported a 67% incidence of grade II-IV articular cartilage lesions at arthroscopy, with the majority found at the medial femoral condyle (49.7%) followed by the patella (33.1%)[64]. This pattern of involvement is reinforced in all available studies. Overall there was a low incidence in the development of meniscal tears following non-operative management. Shelbourne *et al.* reported that 3% of subjects with isolated PCL injuries had meniscal tears on MRI at the time of injury and at 5-year follow-up a further 2% had developed meniscal tears[50]. Similarly Keller *et al.* also reported a low 5% incidence of meniscal tears following non-operative treatment of isolated PCL injuries[47]. Both studies had a higher incidence of medial meniscal injury compared with the lateral meniscus. The larger curvature radius of the medial femoral condyle means that with relative anterior subluxation of the femur; a higher pressure point with less congruence occurs at the tibiofemoral joint entrapping the anterior portion of the medial meniscus[50]. The pattern of the articular cartilage degeneration and meniscal injury correlates with the abnormal kinematics and contact pressures in the medial compartment of the PCL deficient knee demonstrated in cadaveric studies as outlined above[6, 45].

Table 1-4: Muscle Strength

| **Outcome Measure** | **Principle Author** | **LOE** | **Significant Results** |
|---|---|---|---|
| Muscle Strength | Cain[62] | IV | A PCL-d patient developed stronger quadriceps strength in the injured knee (252 ft-lb vs. 240 ft-lb). |
| | Fowler[13] | II | As measured by Cybex dynamometer, there was no significant difference in strength |

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| | | between knees. |
| Hooper[56] | III | Significantly decreased isokinetic knee flexion torque between PCL-d knees, uninjured knees and controls. |
| Keller[47] | IV | As measured by a Cybex dynamometer, the mean score on isokinetic testing was 99% compared with the uninjured knee. |
| MacLean[66] | III | As measured by a Kinetic Communicator isokinetic dynamometer, quadriceps concentric contraction was not significantly different between injured knees and uninjured knees. Quadriceps eccentric/concentric ratio was 1.08 in PCL-d knees and 1.07 in the uninjured knee. Hamstring/quadriceps ratio was less than 0.60 There were significant differences between PCL-d and uninjured eccentric H/Q ratios. |
| Parolie[48] | IV | As measured by a Cybex dynamometer, the mean quadriceps strength of the PCL-d knee was at least 97.7% compared with the uninjured knee. |
| Shelbourne[49] | II | As measured by a Cybex dynamometer, the mean quadriceps strength of the PCL-d knee was 94% compared with the uninjured knee. |

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|---|---|---|
| Shirakura[67] | III | Significantly decreased isokinetic knee flexion torque between PCL-d knees and uninjured knees above $36^0$. |
| Tibone[61] | III | Significantly decreased isokinetic knee extension torque (20%) in PCL-d knees compared with uninjured knees. Significantly decreased mean isometric knee extension force between injured (11.9 kg) compared with uninjured (14.7 kg). |

LOE: Level of Evidence

There are a variety of findings on the effect of PCL deficiency on muscle strength around the knee joint (Table 1-4). Shirakura *et al.* reported in flexion angles greater than $36^0$ there was a significant decrease for quadriceps eccentric and concentric torque in PCL-d knees[67]. Skirakura reported an eccentric/concentric ratio of 1.29 in uninjured knees of 19 patients, 13 of which underwent surgical management. MacLean *et al.* demonstrated that the injured limb was weaker eccentrically for both quadriceps and hamstrings and there was a significant lower hamstrings eccentric/concentric ratio[66]. Hooper *et al.* also found decreased peak extensor torque in subjects with PCL deficiency in both PCL-d and uninjured knees compared with healthy controls. Authors remark that this bilateral decrease in eccentric contraction may reflect patient reluctance to engage in activity after injury when treated with non-operative management. Furthermore, Tibone *et al.* showed that subjects with PCL deficiency demonstrate significantly lower quadriceps peak torque and extension force in their PCL-d knees during isometric and isokinetic testing at $60^0$/second but

not $120^0$/second[61]. Authors concluded that this may be due to decreased mechanical advantage with a shortened movement arm of the extensor mechanism with a posteriorly subluxed tibia. Although studies generally showed that patients had decreased torque, following a period of non-operative rehabilitation four prospective studies found no deficit in quadriceps strength between PCL-d and uninjured knees for subjects with isolated PCL injuries[13, 47-49].

Table 1-5: Proprioception

| Outcome Measure | Principle Author | LOE | Significant Results |
|---|---|---|---|
| Proprioception | Clark[68] | II | The threshold to perception of passive movement was significantly increased in the PCL-d knee (1.19°) compared to the uninjured knee (0.93°). |
| | Safran[69] | III | The threshold to perception of passive movement was significantly increased in the PCL-d knee (1.5°) compared to the uninjured knee (1.2°) at 45° starting point.<br>The threshold to perception of passive movement was significantly increased in the PCL-d knee (1.38°) compared to the uninjured knee (1.29°) at 110° starting point.<br>The ability to reproduce passive |

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| | | | positioning was significantly worse in the PCL-d knee (2.3°) compared with the uninjured knee (3.1°) at a true angle of 110°. The ability to reproduce passive positioning was not significantly different between knees at a true angle of 45°. |

LOE: Level of Evidence

There are two studies that suggest that PCL injury reduces proprioception in the injured limb (Table 1-5). Clark *et al.* demonstrated that at random flexion and extension movements at 30 and $45^0$ the PCL-d knee had a significant increase in the threshold to perception of passive movement compared to the uninjured side[68]. Safran *et al.* reported a significant increase in the threshold at $45^0$ and at $110^0$, but interestingly no significant differences in the ability to reproduce the position angle at $110^0$ [69]. However, it is not known whether loss of proprioception correlates with poorer function.

Table 1-6: Rehabilitation Outcomes

| **Outcome Measure** | **Principle Author** | **LOE** | **Significant Results** |
|---|---|---|---|
| Rehabilitation | Cross[70] | IV | 80% of patients treated conservatively had an excellent or good result as assessed by the authors. |

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| Fowler[13] | II | 100% of patients returned to previous athletic activities without disability. |
| Parolie[48] | IV | 68% returned to previous athletic activities without disability. |
| Shelbourne[49] | II | 50% of patients returned to previous athletic activities without disability. |
| Shelbourne[50] | II | 76% of patients returned to previous athletic activities without disability. |
| Toritsuka[71] | IV | 88% of patients returned to previous athletic activities without disability. |

LOE: Level of Evidence

Review of the literature suggests that the majority of subjects with isolated PCL injuries treated with a non-operative rehabilitation programme return to activity at the same or similar level (Table 1-6). Shelbourne *et al.* reported on 133 subjects 50% of whom were able to make a full return after physical therapy while a further 32% were able to return but at a lower level[49]. In their 6 year follow up study they reported that 76% of 271 subjects were able to return to sport or activity at a similar level[50]. Fowler *et al.* reported that 100% of patients in their prospective trial returned to their previous athletic activities without disability.[13] In addition, Cross and Powell reported over 80% of patients treated conservatively had an excellent or good result on clinical examination. When comparing the mechanism of injury, Cross and Powell reported 85% of sport related injuries achieved a good result compared to only 8% of motor vehicle accidents[70]. Overall, the authors concluded that return to function depended

upon good quadriceps tone and the severity of the original injury. However one case series had disparate conclusions. Toritsuka *et al.* reported in rugby players that although 88% returned to pre-injury levels, 64% had residual limitations in high speed running[71].

Table 1-7:Relationship between Laxity and Functional Outcome

| Outcome Measure | Principle Author | LOE | Significant Results |
|---|---|---|---|
| Laxity | Boynton[46] | III | Significant relationship between degree of posterior laxity and function as measured by a questionnaire developed at the Cincinnati Sports medicine and Orthopaedic Centre. Mean knee score for 1+ posterior drawer was 85.2 whilst 3+ posterior drawer was 61.9. No statistical relationship between degree of laxity and stair walking function. |
| | Dandy[72] | IV | No statistical relationship between degree of posterior laxity and function as measured by clinical examination and a goniometer. |
| | Fontbote[55] | II | Significantly greater instrumented laxity ((6.3 ± 2.0 to 1.4 ± 0.5 mm) |
| | Fowler[13] | II | No relationship between degree of posterior laxity and function as measured by clinical examination and KT-1000 Arthrometer. |
| | Keller[47] | IV | Significant relationship between degree of |

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| | | posterior laxity and function as measured by the Noyes knee scores. 1+: 81, 2+: 72 3+: 58. |
| Parolie[48] | IV | No statistical relationship between degree of instability and function as measured by clinical examination and KT-1000 Arthrometer. |
| Shelbourne[49] | II | No statistical relationship between degree of posterior laxity and function as measured by a KT-1000 Arthrometer, stress radiography and Noyes knee score. |
| Shelbourne[73] | II | No statistical relationship between degree of posterior laxity and function as measured by the Noyes knee score and an International Knee Documentation Committee knee survey.<br>No statistical relationship between degree of laxity and function. |

LOE: Level of Evidence

The correlation between laxity due to PCL injury and its functional consequence is not clearly defined in the literature (Table 1-7). There are many prospective studies that have found no correlation between functional outcomes and posterior laxity in subjects with isolated PCL injuries[48-50, 72-73]. In contrast, Keller et al found that increased laxity resulted in lower functional outcomes with subjects less likely to

return to their pre-injury level of activity[47]. Boynton and Tietjens were able to establish a correlation between the degree of posterior laxity and questionnaire scores for knee functionality and ability to walk[46]. One possible reason to account for this discrepancy is that Keller and Boynton included subjects with chronic PCL deficiency in their studies. Keller evaluated subjects at a mean of 6 years from injury and Boynton at a mean of 13 years. Although both reported increased articular degeneration as time from injury increased, only in the case of Keller *et al.* was this statistically significant. As a result, we are not able to conclude whether the lower functional knee scores over time can be attributed to the increased incidence of articular degeneration.

## Conclusions

Understanding the kinematics of PCL deficiency and its related structures is important for diagnosis and management. The PCL in addition to the posterolateral structures, posterolateral capsule and meniscofemoral ligaments provide important primary and secondary restraints to posterior translation of the medial tibia. Injury to the PCL alters the articulation pattern of the medial compartment and increases the contact pressures within it[6].

Cadaveric studies have shown a strong influence of the PCL as a primary restraint to posterior translation of the tibia with this role peaking at $90^0$. This primary restraint role is confirmed by the clinical examination of the posterior sag test where the tibia subluxes posteriorly with the knee rested at $90^0$. Further research into the area should

include further in-vitro studies that analyse sagittal and coronal plane articulation of the PCL deficient knee. Using MRI as part of the dynamic model that simulates squatting and other sporting activities may help in the analysis. Further, the specific analysis and definition of acute and chronic PCL injuries in terms of articulation patterns may help explain compensatory mechanisms that allow some patients to function with little objective and subjective deficit. Following on from this, the effectiveness of early quadriceps activation training on kinematics associated with PCL injury could be important to inform current rehabilitation protocols.

The current quality of in-vivo studies is low, with few prospective comparative trials. There is high degree of heterogeneity in the patient groups, most being a mixture of acute and chronic PCL-d knees, with different grades of laxity, therefore making direct comparison difficult. However there are some trends that can be delineated.

Gait-analysis studies suggest that despite having a PCL-d knee, there is little difference in kinematics for self-paced activities and only more physically demanding activities highlight more significant compensatory differences. Even in settings where the PCL would normally be under stress, modifications in gait and isokinetic muscle contractions minimise the impact of the PCL deficiency quite successfully. Overall the literature confirms early activation of the gastrocnemius-soleus complex in particular as being the major muscular compensatory mechanism.

Views on the impact of non-operative management of PCL injuries on long-term outcomes are not clear. Some studies suggest that non-operative management is associated with an increased incidence of degenerative arthritis. Longer-term studies need to be performed with operative control groups to allow conclusions regarding

whether observed degenerative changes may be affected by operative interventions such as meniscectomy and altered mechanics after reconstruction. When degenerative changes do occur, they tend to occur in the medial compartments, which correlate with findings from cadaveric studies.

The literature tends to have consistent evidence regarding quadriceps muscle strength. Most studies conclude that peak extensor torque is decreased in PCL-d subjects, however, with adequate rehabilitation; quadriceps strengthening may overcome any such deficits in function. Literature largely reflects an overall conclusion that aggressive rehabilitation is an integral component of non-operative management of isolated PCL injuries. However, considering the younger ages and the potential to develop accelerated degenerative changes long-term, further studies should focus on the long-term comparative outcome of operative and non-operative management of PCL injuries.

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# Chapter Two: Magnetic Resonance Imaging Study Of Alteration Of Tibiofemoral Joint Articulation After Posterior Cruciate Ligament Injury

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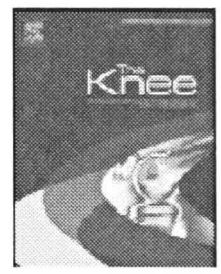


## Magnetic resonance imaging study of alteration of tibiofemoral joint articulation after posterior cruciate ligament injury

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ABSTRACT

Cadaveric studies have shown that the posterior cruciate ligament (PCL) is an important constraint to posterior translation of the tibia. Arthroscopic studies have shown that chronic PCL injuries predispose to articular cartilage lesions in the medial compartment and the patellofemoral joint. The aim of the present study was to investigate sagittal plane articulation of the tibiofemoral joint of subjects with an isolated PCL injury.

Magnetic resonance was used to generate sagittal images of 10 healthy knees and 10 knees with isolated PCL injuries. The subjects performed a supine leg press against a 150 N load. Images were generated at 15° intervals as the knee flexed from 0 to 90°. The tibiofemoral contact and the flexion facet centre (FFC) were measured from the posterior tibial cortex.

The contact pattern and FFC was significantly more anterior in the injured knee from 45 to 90° of knee flexion in the medial compartment compared to the healthy knee. The greatest difference between the mean TFC points of both groups occurred at 75 and 90°, the difference being 4 mm and 5 mm respectively. The greatest difference between the mean FFC of both groups occurred at 75° of flexion, which was 3 mm. There was no significant difference in the contact pattern and FFC between the injured and healthy knees in the lateral compartment.

Our findings show that there is a significant difference in the medial compartment sagittal plane articulation of the tibiofemoral joint in subjects with an isolated PCL injury.



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## Abstract

Cadaveric studies have shown that the posterior cruciate ligament (PCL) is an important constraint to posterior translation of the tibia. Arthroscopic studies have shown that chronic PCL injuries predispose to articular cartilage lesions in the medial compartment and the patellofemoral joint. The aim of the present study was to investigate sagittal plane articulation of the tibiofemoral joint of subjects with an isolated PCL injury.

Magnetic resonance was used to generate sagittal images of 10 knees of healthy controls and 10 knees with isolated PCL injuries. The subjects performed a supine leg press against a 150N load. Images were generated at 15 degree intervals as the knee flexed from 0 to $90^0$. The tibiofemoral contact (TFC) and the flexion facet centre (FFC) were measured from the posterior tibial cortex.

The TFC and FFC were significantly more anterior in the injured knee from 45 to $90^0$ of knee flexion in the medial compartment compared to the knees of healthy controls. The greatest difference between the mean TFC points of both groups occurred at 75 and $90^0$, the difference being 4 mm and 5 mm respectively. The greatest difference between the mean FFC of both groups occurred at $75^0$ of flexion, which was 3 mm. There was no significant difference in the contact pattern and FFC between the injured and knees of healthy controls in the lateral compartment.

Our findings show that there is a significant difference in the medial compartment sagittal plane articulation of the tibiofemoral joint in subjects with an isolated PCL injury.

KEYWORDS

Posterior cruciate ligament, Posterior cruciate ligament injury, Tibiofemoral joint sagittal articulation,

## Introduction

Biomechanical studies show that the posterior cruciate ligament (PCL) is the most important constraint to posterior translation of the tibia above $30^0$ of knee flexion[1-2]. At flexion angles below this, the posterolateral complex is the most important restraint to posterior tibial translation[3]. The PCL is most commonly injured when a direct blow to the flexed knee pushes the tibia posteriorly relative to the femur[4]. In the literature, there is no clear consensus on the indications and benefits of surgical reconstruction of an injured PCL. PCL injuries are most often managed non-operatively with physiotherapy. Following non-operative management only a minority of subjects have persistent instability symptoms but in the elite sporting population the majority of subjects are unable to compete at preinjury level[5]. Recent arthroscopic studies have suggested that PCL injury does not have a benign natural history but may predispose to articular cartilage damage[6-7]. In one study, arthroscopic evaluation of 181 subjects with a 5 year history of an isolated PCL injury, 80% of subjects had articular cartilage damage to the medial femoral condyle and 50% to the patella[8]. The aim of this study is to use magnetic resonance imaging (MRI) to determine the effects of PCL injury on the sagittal plane articulation of the tibiofemoral joint. The hypothesis of the study is that PCL injury would alter the sagittal plane articulation of the tibiofemoral joint.

# Methods

## Study design

This is a case control study that uses a MRI model, previously described to study ACL injuries[9-12], to study the effects of PCL injury on sagittal plane articulation of the tibiofemoral joint. The tibiofemoral contact (TFC) point and the position of the femoral flexion facet centre (FFC) were chosen as the variables to measure the sagittal plane motion of the tibiofemoral joint. The TFC point allows assessment of tibial sagittal anterior and posterior translation as the knee flexes from $0\text{-}90^0$ and the FFC similarly allows assessment of femoral motion. As the femoral articular surface is curved the FFC allows assessment of not only translation but also glide. Most importantly Scarvell et al. have demonstrated that differences between TFC and FFC positions with knee flexion between healthy and injured knees occurs with pathology[11].

## Subject selection

Twenty subjects participated in the study. Ten subjects with no history of knee complaints and normal clinical examination were used as controls. The control subjects were aged between 26 and 39 years. There were five females and five male subjects. Ten subjects with unilateral PCL injuries were recruited for the study. The subjects were recruited from local orthopaedic and sport medicine practices. The subjects were aged between 18 and 47 years. There were five females and five males. An attempt was made to select controls matched by sex and age. Isolated PCL injury

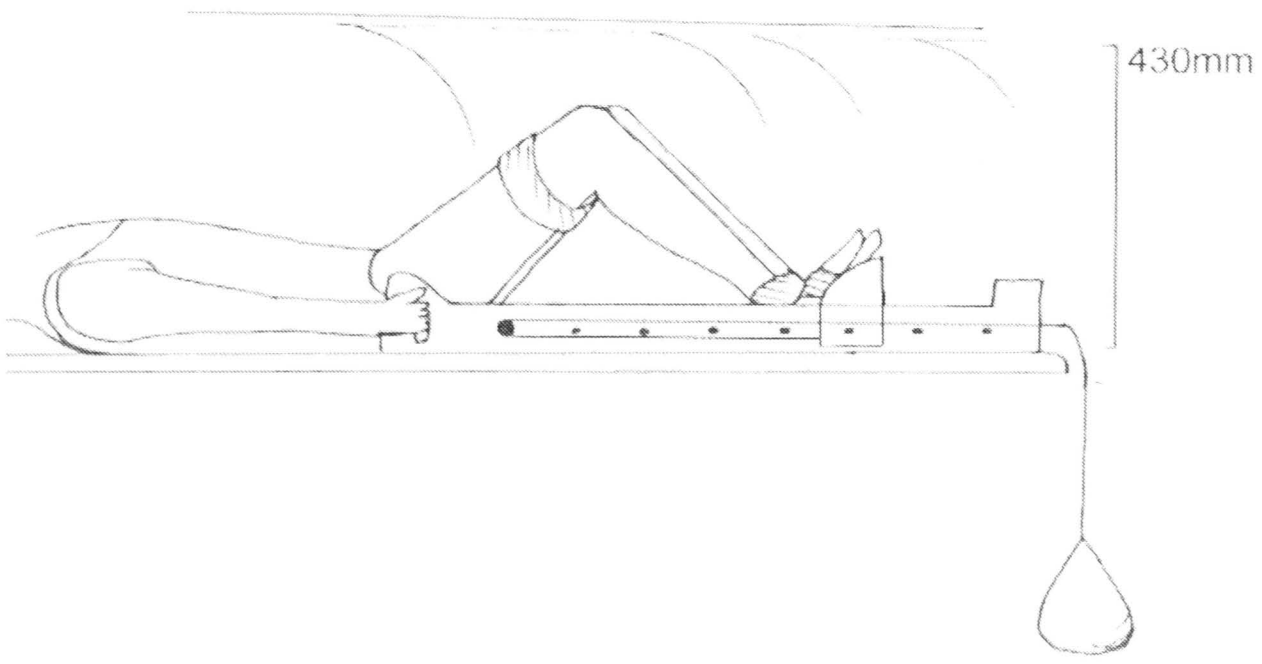
was diagnosed on clinical examination and MRI. On clinical examination PCL injury was suggested by posterior sag and posterior draw test. The dial test was used to exclude subjects with concomitant posterolateral corner injuries. The dial test is performed with the patient in the prone position. The thigh-foot angle is measured with an external rotation stress applied at both $30^0$ and $90^0$ to both the injured and uninjured sides. External rotation of the tibia $>10^0$ indicates posterolateral corner injury. Increased external rotation at $30^0$ but not $90^0$ indicates isolated posterolateral corner injury. Increased external rotation at both $30^0$ and $90^0$ indicates combined PLC and PCL injury. Despite the dial test being commonly used to diagnose PLC injuries sensitivity and specificity values have not been reported for the test[29]. Subjects were excluded if there were any contraindications to MRI, may have been pregnant, or if they were over 180cm tall (to permit knee flexion in the MRI tunnel). PCL injuries were sustained from a time period of 3 months to 21 years before the study examination. Nine subjects sustained the injury through sports (four from netball, four from rugby and one from soccer) and one subject sustained the injury outside of sport. All subjects provided informed consent. Normal knees were used as controls rather than the healthy contralateral knees because anatomical variations in tibial slope have been identified as risk factors for anterior cruciate ligament injury[13]. Ethics approval for the study was obtained from the ACT Health and Australian National University Human Research Ethics Committees.

## MRI imaging procedure

Subjects performed a supine leg press between 0 and $90^0$ on a wooden frame with a sliding footplate fitted to the MRI couch. Isometric contractions at 15 degrees

intervals were achieved by the use of a leg press weighted by a 150N load via a rope and pulley to resist leg extension and thereby simulate a weight bearing squat (Figure 2-1). Elastic straps stabilised the thighs, feet and ankles. Imaging of both knees simultaneously was performed. Parasagittal images perpendicular to the tibial plateau were generated through each knee

Figure 2-1: Subjects' position in the MRI scanning tunnel. The knees were positioned at 15 degree intervals between 0 and $90^0$ flexion, pressing down through the feet against a 150N load

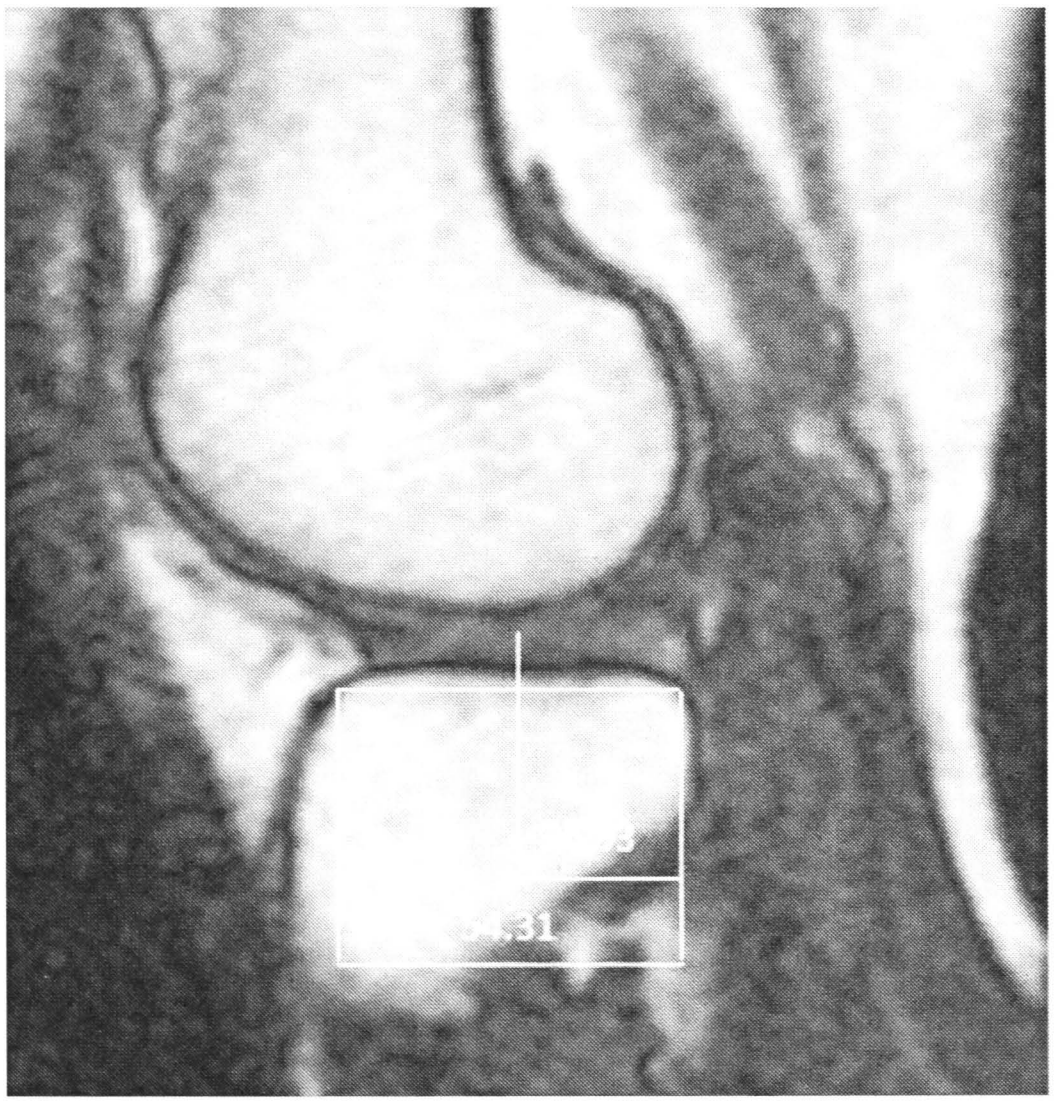



## Tibiofemoral contact point measurement

The position of the tibiofemoral contact (TFC) with the tibial plateau was recorded as the distance from the posterior tibial cortex to the point of the TFC of the medial and lateral femoral condyle (Figure 2-2). The sagittal slice with the greatest anteroposterior diameter of the medial and lateral tibial plateau was selected to perform the measurements. Where contact occurred over a wide area, the area centroid was used. To account for variation in the size of subjects, cortex to contact distance measurements were normalised to a tibial plateau size of 50 mm. The mean

anterior-posterior diameter of the medial tibial plateau was 48 ± 5.4 mm, and the lateral tibial plateau was 41 ± 2.47 mm.

Figure 2-2: Sagittal images through the centre of the compartment of the knee were used to measure the tibiofemoral contact (TFC). The distance was measured through the posterior tibial cortex to the centre of the area of contact.

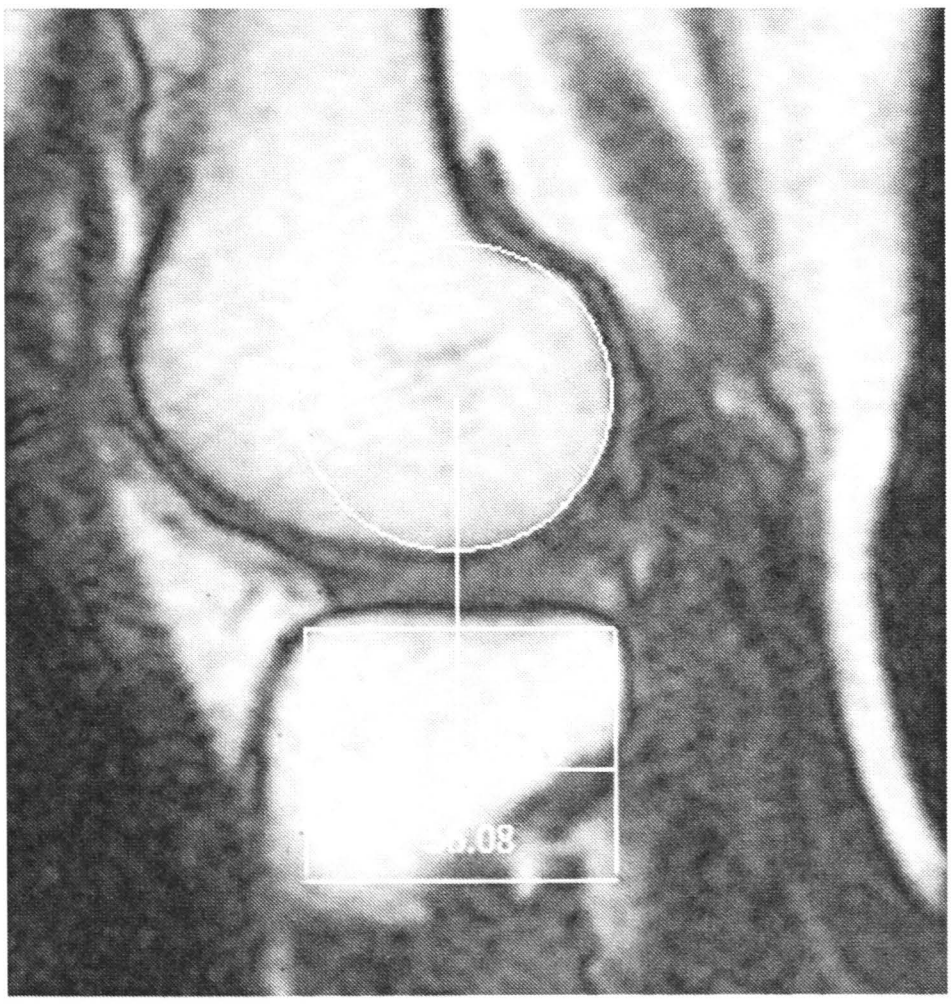


## Flexion facet centre measurement

The position of the flexion facet centre (FFC) over the tibial plateau was located by using a three stage measurement technique with a computer assisted design program (TurboCAD Pro V16.1, D Cheke, IMSI Design) Figure 2-3). First, the FFC was identified by fitting a circle to define the flexion arc of the posterior condyle. This involved using an arc function to identify three points on the posterior aspect of the

femur which could then be incorporated into a circle of bit fit. Second, the tibial plateau was defined by a line from the posterior tibial cortex, parallel to the tibial plateau. Lastly, a line was drawn through the FFC perpendicular to the tibial plateau line to measure the distance from the posterior tibial cortex to the intersection of the perpendicular line. The sagittal slice with the greatest anteroposterior diameter of the medial and lateral tibial plateau was selected to perform the measurements.

Figure 2-3: The position of the flexion facet centre over the tibial plateau was measured in three steps: the arc and centre (FFC) of the posterior femoral condyle were defined, the tibial plateau was defined, and the distance from the perpendicular through the centre to the posterior tibial cortex was determined.



## Precision

The precision of both methods of measurement was tested by repeating measurement from the original scanned images on two occasions at least 24 hours apart. The precision of mapping the contact points for the medial and lateral compartments was very high with intra class correlation 0.95 ( 99% confidence interval was 0.92 0.96). The precision of measuring of the FFC was also very high with intra class correlation of 0.93( 95% confidence interval was 0.88-0.93). The greatest difference observed between the repeated measurements was 0.7 mm for the mapping the TFC point and 0.9 mm for mapping the FFC.

## Statistical Analysis

Statistical analysis was carried out using statistiXL version 1.8 for Microsoft Excel. A two-way repeated measures analysis of variance with Tukey and Scheffe post hoc tests were used to compare the tibiofemoral contact points and FFC positions between the healthy, the PCL deficient knees and the contralateral side. A p value of less than 0.05 was regarded as statistically significant.

## Results

Table 2-1 shows the average and standard deviations for the TFC points and FFC for the healthy and PCL injured knees.

Table 2-1: The position of the tibiofemoral contact point and flexion facet centre in the healthy and PCL injured knees of 20 subjects (mean and standard deviation) measured in millimetres from the posterior tibial cortex.

| | Tibiofemoral contact | | | | Flexion facet centres | | | |
|---|---|---|---|---|---|---|---|---|
| | Healthy | | PCL Injured | | Healthy | | PCL Injured | |
| | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| Lateral Compartment | | | | | | | | |
| 0 | 29 | 2.3 | 30 | 1.0 | 25 | 1.5 | 26 | 1.1 |
| 15 | 27 | 1.8 | 28 | 0.6 | 24 | 1.4 | 24 | 0.7 |
| 30 | 26 | 1.6 | 26 | 1.6 | 22 | 1.6 | 23 | 1.0 |
| 45 | 22 | 1.6 | 22 | 1.1 | 20 | 1.6 | 21 | 0.4 |
| 60 | 19 | 1.8 | 21 | 1.0 | 18 | 1.8 | 19 | 1.4 |
| 75 | 18 | 1.7 | 20 | 0.7 | 17 | 1.7 | 18 | 1.3 |
| 90 | 18 | 1.9 | 19 | 1.3 | 17 | 1.9 | 18 | 1.4 |
| Medial Compartment | | | | | | | | |
| 0 | 31 | 1.3 | 32 | 1.6 | 21 | 1.3 | 23 | 1.6 |
| 15 | 29 | 1.2 | 30 | 1.3 | 23 | 1.2 | 22 | 0.7 |
| 30 | 26 | 1.3 | 27 | 1.4 | 22 | 1.3 | 23 | 0.9 |
| 45 | 24 | 1.4 | 27 | 1.4 | 22 | 1.4 | 24 | 1.4 |

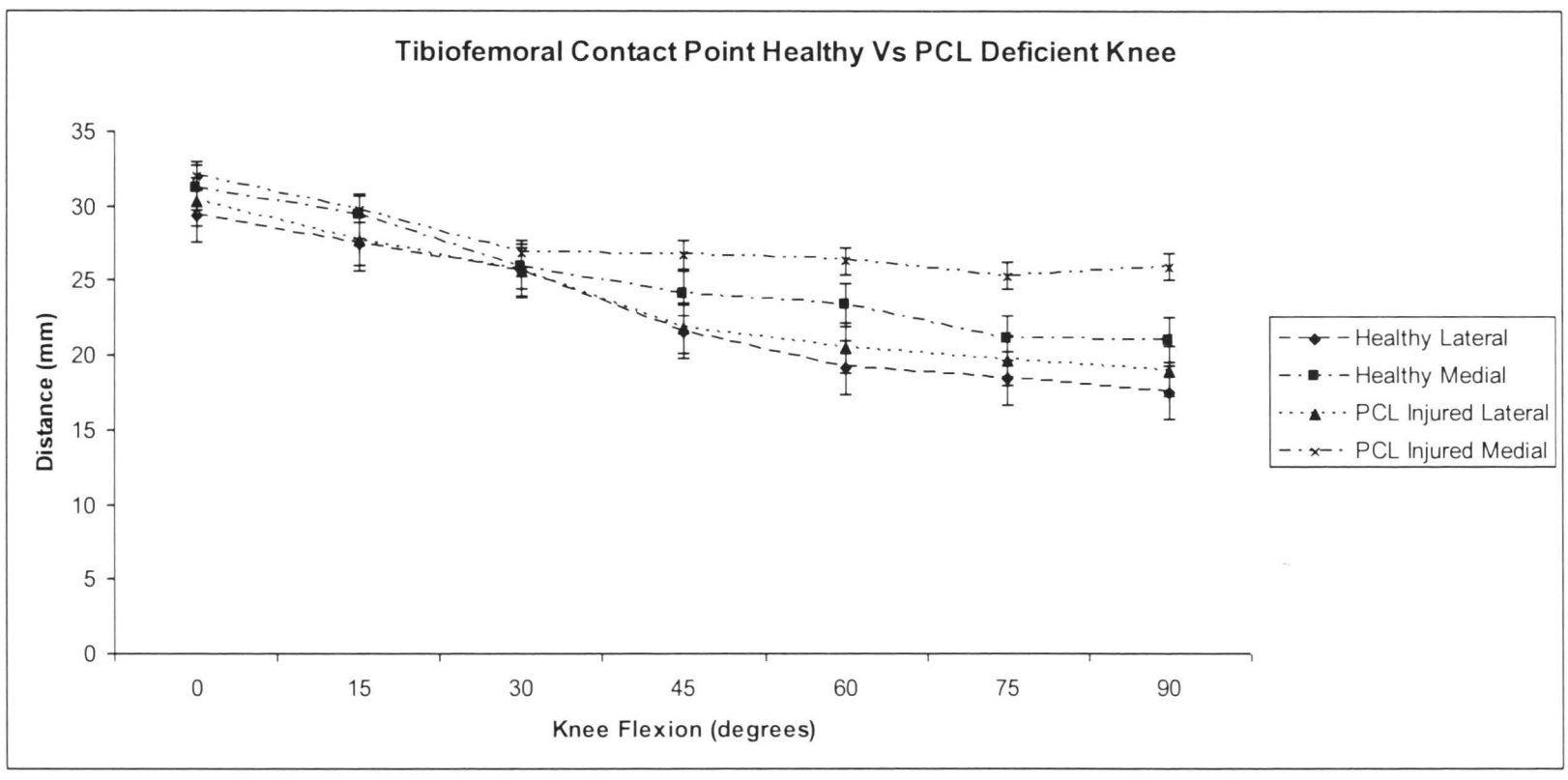
| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 60 | 23 | 1.4 | 26 | 0.6 | 21 | 1.4 | 23 | 0.9 |
| 75 | 21 | 1.4 | 25 | 1.0 | 20 | 1.4 | 23 | 1.8 |
| 90 | 21 | 1.4 | 26 | 0.9 | 21 | 1.4 | 23 | 1.4 |

## Sagittal plane articulation of the tibiofemoral joint in healthy subjects

### Tibiofemoral contact point

In the knees of healthy controls the TFC point moved anterior to posterior as the knee flexed from 0 to $90^0$ (Figure 2-4). In full knee extension the medial compartment had a more anterior mean contact point than the lateral compartment. Between 0 and $30^0$ the mean contact point in the medial compartment moved posteriorly by 5 mm, which was 0.16 mm per degree. Between 0 and $30^0$ the mean contact point in the lateral compartment moved posteriorly by 3 mm, which was 0.10 mm per degree. Between 45 and $90^0$ the mean contact point in the medial compartment did not move posteriorly as much, 3 mm in $45^0$, or 0.07 mm per degree. The mean contact point in the lateral compartment also did not move posteriorly as much 4 mm in $45^0$, or 0.08 mm per degree.

Figure 2-4. Graph comparing tibiofemoral contact points in the healthy and PCL injured knee, performing a leg press against a 150N load through a flexion arc of 0 to $90^0$. The pattern of tibiofemoral contact positions in healthy and PCL injured knees is similar in the lateral compartment. The position of the tibiofemoral contact point is significantly more anterior in the medial compartment above $45^0$ of flexion for the PCL injured group.

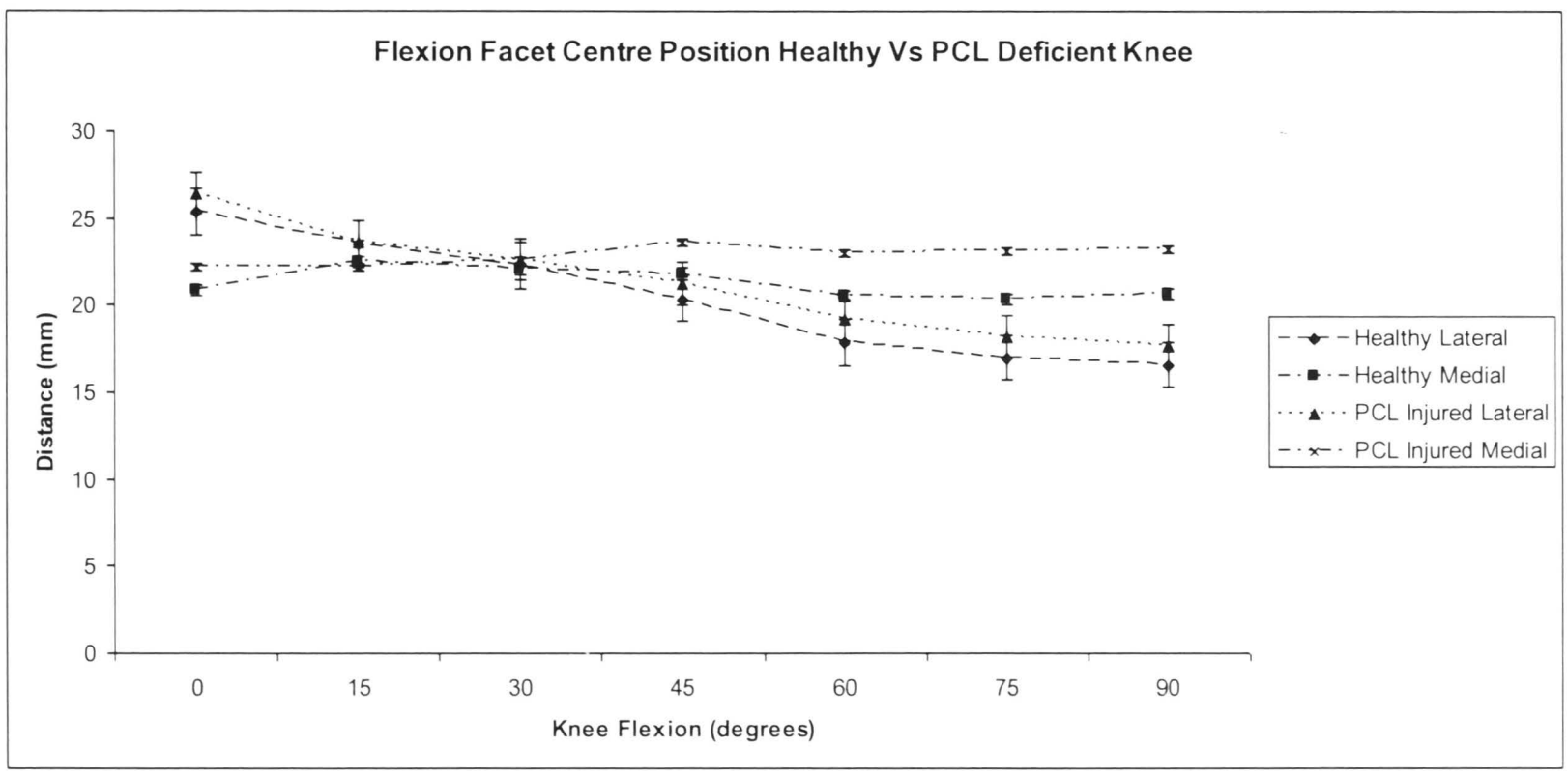



## Flexion facet centre

In the medial compartment the mean FFC was positioned posteriorly over the tibial plateau in knee extension (Figure 2-5). The medial mean FFC moved anteriorly by 2 mm as the knee flexed from 0 to $15^0$. From 30 to $90^0$ the medial mean FFC returned to its posterior position over the medial tibial plateau. During the entire flexion arc form 0 to $90^0$ the medial FFC was positioned over the tibial plateau within a mean distance of 23 mm to 20 mm from the posterior tibial cortex. In the lateral compartment the mean FFC moved posteriorly as the knee flexed from 0 to $90^0$. At

full extension the mean FFC was located over the tibial plateau 25 mm from the posterior tibial cortex. At $90^0$ the mean FFC in the lateral compartment was located over the tibial plateau 17 mm form the posterior tibial cortex. The mean FCC in lateral compartment had moved 8 mm at an average of 0.09 mm per degree.

Figure 2-5. Graph comparing flexion facet centre position in the healthy and PCL injured knee, performing a leg press against a 150N load through a flexion arc of 0 to $90^0$. The position of the flexion facet centre in healthy and PCL injured knees is similar in the lateral compartment. The position of the flexion facet centre is significantly more anterior in the medial compartment above $45^0$ of flexion for the PCL injured group.




## Sagittal plane articulation of the tibiofemoral joint in subjects with an isolated PCL injury

## Tibiofemoral contact point

In the medial compartment of the PCL injured knee the mean TFC point was located more anteriorly on the tibia throughout the flexion arc from 0 to $90^0$ compared to the medial compartment of the knees of healthy controls (Figure 2-4). In extension the mean TFC point was located 32 mm from the posterior tibial cortex. From 0 to $30^0$ the difference in the location of the contact points between the PCL injured and knees of healthy controls were not statistically significant ($p > 0.05$) . The anterior location of medial compartment mean TFC point in the PCL deficient knee was statistically significant from 45 to $90^0$ at all measured points ($p < 0.05$). The greatest difference between the mean TFC points occurred at 75 and $90^0$, the difference being 4 mm and 5 mm respectively. In the lateral compartment the mean TFC point of the PCL injured knees was located more anterior than the mean TFC point of the lateral compartment of the knees of healthy controls throughout the flexion range but there was no statistically significant difference at any of the measured points.

## Flexion facet centre

In the medial compartment of the PCL injured knee the mean FFC was centred more anteriorly on the tibia compared to the mean FFC of the medial compartment of the knees of healthy controls (Figure 2-5). This was not significant at 0, 15 and $30^0$ but statistically significant at 45, 60, 75 and $90^0$. The greatest difference of the FFC occurred at $75^0$ of flexion, which was 3 mm. In the lateral compartment of the PCL deficient knee the FFC was centred more anteriorly than the knees of healthy controls

over the tibial plateau as the knee flexed through 0 to $90^0$, however, there was no statistical difference.

## Sagittal plane articulation of the tibiofemoral joint of the healthy contralateral knee in subjects with an isolated PCL injury

There was no significant difference in the position of both the mean TFC point and mean FFC between the contralateral knees of healthy controls and normal knee in both the medial and lateral compartments throughout the flexion arc from 0 to $90^0$. In the medial compartment the mean TFC and FFC positions were significantly more anterior in the PCL injured knee compared with the contralateral side with the greatest difference replicating the relationship between the normal and PCL deficient knee. Similarly, there was no difference in the lateral compartment in the positions of the mean TFC point and FFC between the PCL deficient knee and the contralateral side.

## Discussion

The aim of this study was to investigate the effect of an isolated PCL injury on the sagittal plane articulation of the tibiofemoral joint. This study demonstrated that PCL deficiency produced a significant change in the sagittal plane articulation of the tibiofemoral joint in the medial compartment between 45 and $90^0$ of knee flexion compared to the knees of healthy controls and contralateral side. Specifically, the TFC point and FFC move more anteriorly as the knee flexes. Further, this study demonstrated that PCL deficiency produced no significant change in the sagittal plane

articulation of the lateral compartment of the tibiofemoral joint. The results are consistent with cadaveric studies which show that the PCL is an important constraint to posterior tibial subluxation and clinical studies which show that chronic PCL deficient knees are associated with medial compartment chondral lesions.

This study used an in vivo model to describe the sagittal plane articulation of the PCL deficient knee. The study incorporated a supine leg press against a 150N load through a flexion arc of 0 to $90^0$ to simulate a squat. The benefits of using this in vivo model over cadaveric sectioning studies is that this model allows the study to replicate the neuromuscular contribution to joint stability that occurs in the clinical setting of PCL injury. This study used knees of healthy controls as the control rather than the contralateral knee as there have been studies that have shown that variations in bony and soft tissue anatomy may predispose to ligamentous injuries of the knee[14-16]. However, the results of this study showed there was no difference in the articulation pattern of the healthy and contralateral knees.

The limitations of this study include supine analysis and small sample size. The supine leg press was intended to simulate a squat. It is difficult to extrapolate whether this replicates the forces during sporting or activities of daily living and as such could potentially be a source of error in our results. Although the number in the study was small (10 knees of healthy controls and 10 PCL injured knees), the number was sufficient to demonstrate a significant difference in the sagittal plane articulation of the PCL deficient knee. Furthermore, in vivo studies on PCL kinematics have used similar or smaller participant numbers[17].

The findings in this study are consistent with results of several cadaveric studies in the literature. Cadaveric studies have shown that isolated cutting of the PCL allows a minimal increase in posterior draw when the knee is extended, and that there is a greater increase in laxity as the knee flexes with the greatest displacement at $90^0$ of knee flexion[18-23]. Veltri et al. demonstrated that the posterolateral structures of the knee exert a maximal posterior tibial restraining force from 0 to $30^0$ of knee flexion[24]. Intact posterolateral structures explain why anterior tibial displacement of the PCL injured knee in our study was not significant between 0 and $30^0$ of knee flexion. In addition, Robinson et al analysed the role of the posteromedial capsule in resisting posterior translation of the tibia. They demonstrated that the posteromedial capsule controlled valgus, internal rotation, and posterior drawer in extension, resisting 42% of a 150-N drawer force when the tibia was in internal rotation. Their finding may also explain why PCL injury did not produce significant anterior displacement of the tibia at less than $30^0$ of knee flexion in our study. However, there have been no cadaveric studies that have assessed the relative contributions of the medial and lateral compartments of the knee to posterior translation.

There have been very few in vivo studies investigating the sagittal plane articulation of the PCL deficient knee. Logan et al analysed the FFC in 6 subjects with isolated PCL rupture undergoing a weight bearing squat using open access MRI[17]. They reported significant posterior subluxation of the medial tibia at 0, 20, 45 and $90^0$ of knee flexion. In addition, they reported that the sagittal plane articulation of the lateral compartment was not altered by posterior cruciate ligament rupture. Our results are in agreement with Logan et al which demonstrate altered articulation in the

medial compartment. One point of difference is that Logan et al noted significant altered articulation throughout the arc of flexion from to 0 to $90^0$ whereas we found that articulation was significantly different from 45 to $90^0$. There are two possible explanations for this discrepancy. Firstly, it is not clear whether Logan et al excluded subjects with concomitant posterolateral corner injury as cadaveric studies have shown that posterolateral corner injuries are most important constraint to posterior tibial translation from 0 to $30^{024}$. The weight bearing model of Logan et al is a more functional model of testing the kinematics of PCL deficiency than our model in which the subject is supine. The increased forces with weight bearing may accentuate posterior tibial subluxation in the PCL deficient knee. It is also important to note that in our study we used two methods, the TFC point and FFC, to assess knee kinematics whereas Logan et al had only used the FFC. Both methods in our study had concurring findings. Castle et al used stress radiography to assess the effects of PCL rupture on knee articulation at 35 and $70^0$ of flexion[25]. They showed that at $70^0$ of flexion the tibia is subluxed posteriorly by 6 mm and no difference at $35^0$ of flexion. They did not analyse the relative contributions of the medial and lateral sides of the knee to posterior tibial translation but their results are nevertheless in agreement with our study. Donell et al published on a pilot study that compared tibiofemoral articulation of five PCL injured subjects with four normal subjects who underwent MRI with a novel splint that stresses the tibia in a posterior direction[26]. They reported that there was no marked posterior tibial translation for the PCL injured knee in posterior drawer compared to the normal knee. Possible reasons why their findings may differ from our study are the effects of secondary constraint from the posterolateral structures, the increased force on the tibia with a supine leg press

compared to a static drawer and the small number and therefore power of their study. Unfortunately, their follow up study only reported on ACL, not PCL rupture[27].

The findings of our study complement those of arthroscopic studies that have shown that chronic rupture of the PCL leads to increased incidence of chondral lesions on the medial femoral condyle. Strobel et al arthroscopically evaluated subjects with a more than 5 year history of a symptomatic PCL injury. They reported that 80% of subjects had an articular lesion of the medial femoral condyle and 50% had articular lesions at the patellofemoral articulation[8]. These findings complement those of our study which show that medial compartment is site of abnormal articulation in the PCL deficient knee. Cadaveric studies have complemented these findings by showing that isolated PCL sectioning increases the articular contact pressure within the medial compartment of the knee[28].

In conclusion, there is significant posterior subluxation of the medial tibial plateau from 45 to $90^0$ in the PCL deficient knee. Physiotherapy and operative interventions should therefore aim to address the altered sagittal plane articulation in the medial compartment of the PCL deficient knee.

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# Chapter Three: Sagittal Plane Articulation of The Contralateral Knee of Subjects With Posterior Cruciate Ligament Deficiency: An Observational Study

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RESEARCH ARTICLE Open Access

## Sagittal plane articulation of the contralateral knee of subjects with posterior cruciate ligament deficiency: an observational study

Sivashankar Chandrasekaran[1*], Jennifer M Scarvell[1], Graham Buirski[2], Kevin R Woods[1] and Paul N Smith[1]

**Abstract**

**Background:** The aim of the present study was to compare the in vivo articulation of the healthy knee to the contralateral knee of subjects with acute and chronic PCL injuries.

**Methods:** Magnetic resonance was used to generate sagittal images of 10 healthy knees and 10 knees with isolated PCL injuries (5 acute and 5 chronic). The subjects performed a supine leg press against a 150 N load. Images were generated at 15 degree intervals as the knee flexed from 0 to 90 degrees. The tibiofemoral contact (TFC), and the centre of the femoral condyle (as defined by the flexion facet centre (FFC)), were measured from the posterior tibial cortex.

**Results:** There was no significant difference in the TFC and FFC between the healthy knee and contralateral knee of subjects with acute and chronic PCL injuries in the medial and lateral compartments of the knee.

**Conclusions:** The findings of this study suggest there is no predisposing articulation abnormality to PCL injury; in the setting of chronic injury the contralateral knee does not modify its articulation profile and the contralateral knee can be used as a valid control when evaluating the articulation of the PCL deficient knee.

**Keywords:** Posterior cruciate ligament, Posterior cruciate ligament injury, Knee articulation, Contralateral knee in posterior cruciate ligament injury, Risk factors for posterior ligament injury

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## Abstract

Background: The aim of the present study was to compare the in vivo articulation of the knees of healthy controls to the contralateral knee of subjects with acute and chronic PCL injuries.

Methods: Magnetic resonance was used to generate sagittal images of ten knees of healthy controls and ten knees with isolated PCL injuries (five acute and five chronic). The subjects performed a supine leg press against a 150N load. Images were generated at 15 degree intervals as the knee flexed from 0 to $90^0$. The tibiofemoral contact (TFC), and the centre of the femoral condyle (as defined by the flexion facet centre (FFC)), were measured from the posterior tibial cortex.

Results: There was no significant difference in the TFC and FFC between the knees of healthy controls and contralateral knee of subjects with acute and chronic PCL injuries in the medial and lateral compartments of the knee.

Conclusions: The findings of this study demonstrate that the contralateral knee can be used as a valid control when evaluating the articulation of the PCL deficient knee.



KEYWORDS

Posterior cruciate ligament, Posterior cruciate ligament injury, Knee articulation, Contralateral knee in posterior cruciate ligament injury, Risk factors for posterior ligament injury

## Background

Cadaveric studies demonstrate that the posterior cruciate ligament (PCL) is the most important constraint to posterior translation of the tibia above $30^0$ of knee flexion[1-2]. This finding has been supported by in vivo studies that have demonstrated significant posterior translation of the medial tibia in subjects with PCL injuries as the knee flexes from 0 to $90^0$ in comparison to the contralateral side[3]. However, it is debateable whether the contralateral knee can be used as a valid normal control because anatomical variations such as narrower intercondylar notch anatomy and variation in tibial slope have been identified as risk factors for anterior cruciate ligament injury[4]. The cruciate ligaments provide important proprioceptive feedback about knee stability[5]. It has not been investigated whether the contralateral knee in a subject with a PCL injury undergoes any adaptive changes as a result of the abnormal articulation pattern in the injured knee. The aim of this study is to investigate whether the contralateral knee in subjects with acute and chronic PCL injuries can be used as a valid healthy control and whether the articulation pattern in the contralateral knee of chronic PCL deficient subjects exhibits adaptive articulation patterns.

## Methods

### Study design

This is a case control study that uses a MRI model, previously described to study ACL injuries[6-9], to study the sagittal plane articulation of the tibiofemoral joint of the contralateral knee of subjects with PCL deficiency.

## Subject selection

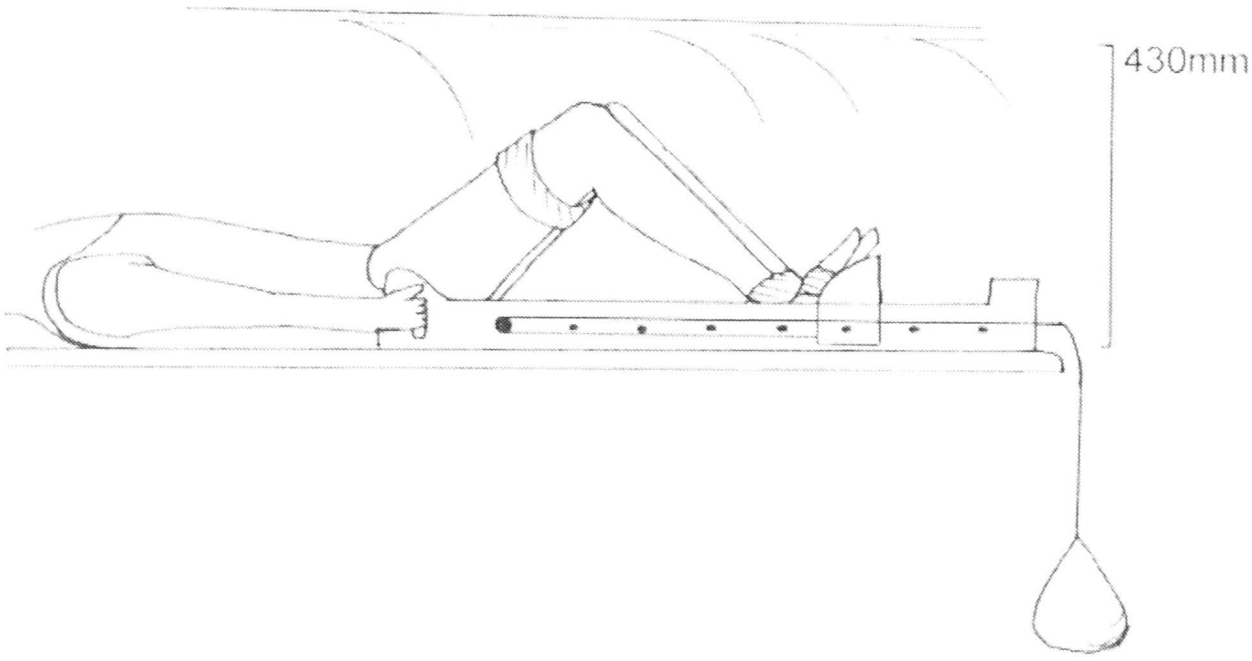
Twenty subjects participated in the study. Ten subjects with no history of knee complaints and normal clinical examination were used as controls. The control subjects were aged between 26 and 39 years. There were five females and five male subjects. Ten subjects with unilateral PCL injuries were recruited for the study. Isolated PCL injury was diagnosed on clinical examination and MRI. On clinical examination PCL injury was suggested by posterior sag and posterior draw test. The dial test was used to exclude subjects with concomitant posterolateral corner injuries. Subjects were excluded if there were any contraindications to MRI, may have been pregnant, or if they were over 180cm tall (to permit knee flexion in the MRI tunnel). Five subjects had an acute PCL injury and five subjects had a chronic PCL injury. An acute PCL injury was defined as the persistence of bone bruising on diagnostic MRI at the time the study was undertaken. At the time the study was undertaken, the time since injury ranged from two to nine weeks. There were two females and three males. The age of these subjects ranged from 18 to 27 years. Four of the subjects had a knee brace locked in extension and had not began physiotherapy exercises and one subject was three weeks from removal of the brace and had begun quadriceps strengthening exercises. All subjects had an effusion on examination, decreased range of motion compared to the contralateral knee but no patellofemoral crepitus. Four subjects sustained the injury through sports (one from netball, one from soccer and two from

rugby) and one subject outside of sport (sustained injury whilst falling from a two metre height). The age of the subjects with a chronic PCL injury ranged from 39 to 47 years. There were three females and two males. PCL injuries were sustained from a time period of 5 to 21 years. No subject had bone bruising on MRI at the time of the study and no effusion on clinical examination. All subjects complained of no symptoms from their knee during activities of daily living. All subjects were able to bicycle with their knee injury. All subjects in the chronic PCL group sustained the injury whilst playing sport (two from rugby and three from netball). Clinically the chronic PCL injury group were examined for evidence of degenerative joint disease. None of the subjects demonstrated joint line tenderness or had reduced range of motion compared to the contralateral side. However, all of the subjects had patellofemoral joint crepitus. All subjects provided informed consent. Ethics approval for the study was obtained from the Department of Health and university human research ethics committees.

## MRI imaging procedure

Subjects performed a supine leg press between 0 and $90^0$ on a wooden frame with a sliding footplate fitted to the MRI couch. The leg press was weighted by a 150N load via a rope and pulley to resist leg extension and thereby simulate a weight bearing squat (Figure 3-1). Elastic straps stabilised the thighs, feet and ankles. Imaging of both knees simultaneously was performed. Parasagittal images perpendicular to the tibial plateau were generated through each knee

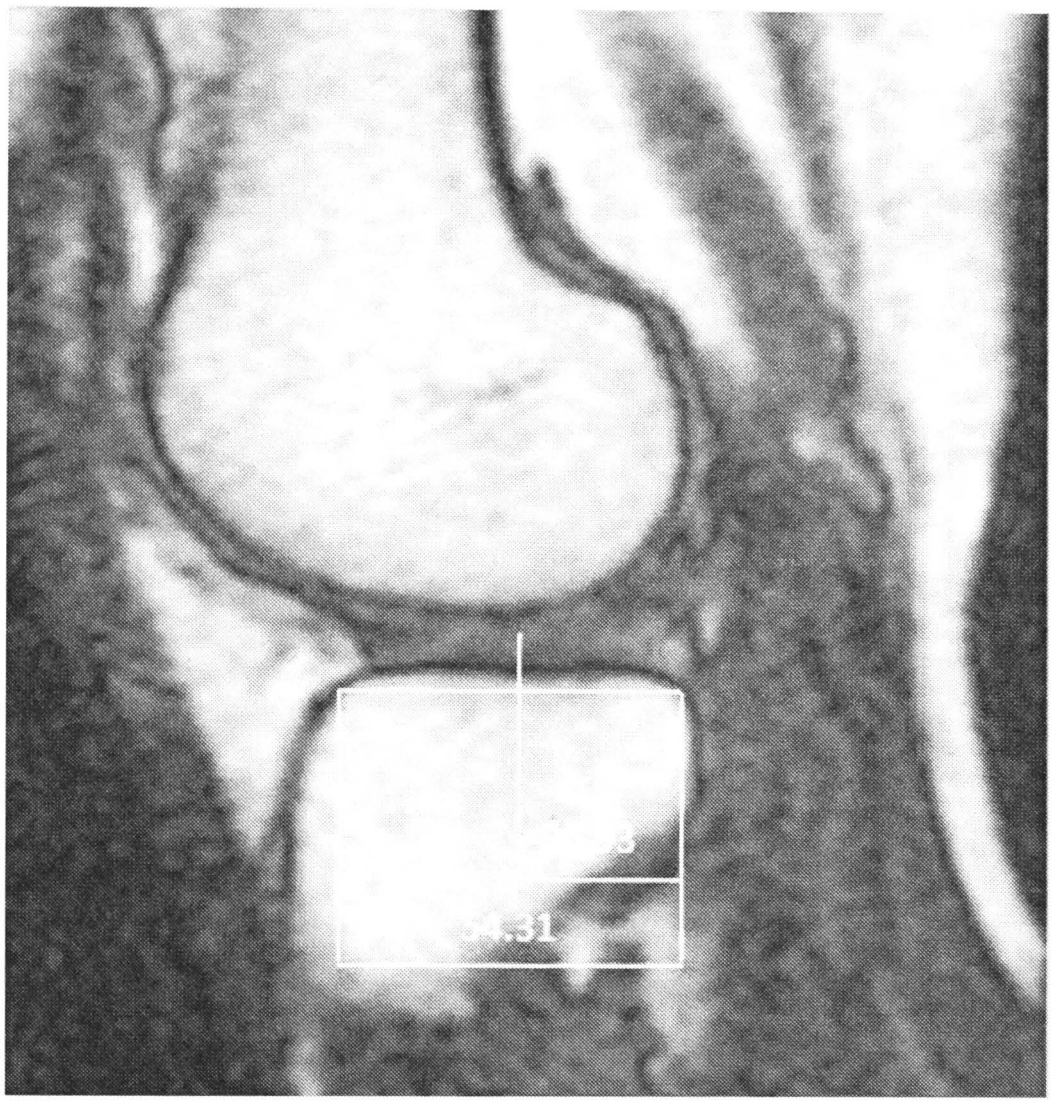
Figure 3-1: Subject's position in the MRI scanning tunnel. The knees were positioned at 15 degree intervals between 0 and $90^0$ flexion, pressing down through the feet against a 150N load.




## Tibiofemoral contact point measurement

The position of the tibiofemoral contact (TFC) with the tibial plateau was recorded as the distance from the posterior tibial cortex to the point of the TFC of the medial and lateral femoral condyle (Figure 3-2). The sagittal slice with the greatest anteroposterior diameter of the medial and lateral tibial plateau was selected to perform the measurements. Where contact occurred over a wide area, the area centroid was used. To account for variation in the size of subjects, cortex to contact distance measurements were normalised to a tibial plateau size of 50 mm. The mean anterior-posterior diameter of the medial tibial plateau was 48 ± 5.4 mm, and the lateral tibial plateau was 41 ± 2.4 mm.

Figure 3-2: Sagittal images through the centre of the compartment of the knee were used to measure the tibiofemoral contact (TFC). The distance was measured through the posterior tibial cortex to the centre of the area of contact.

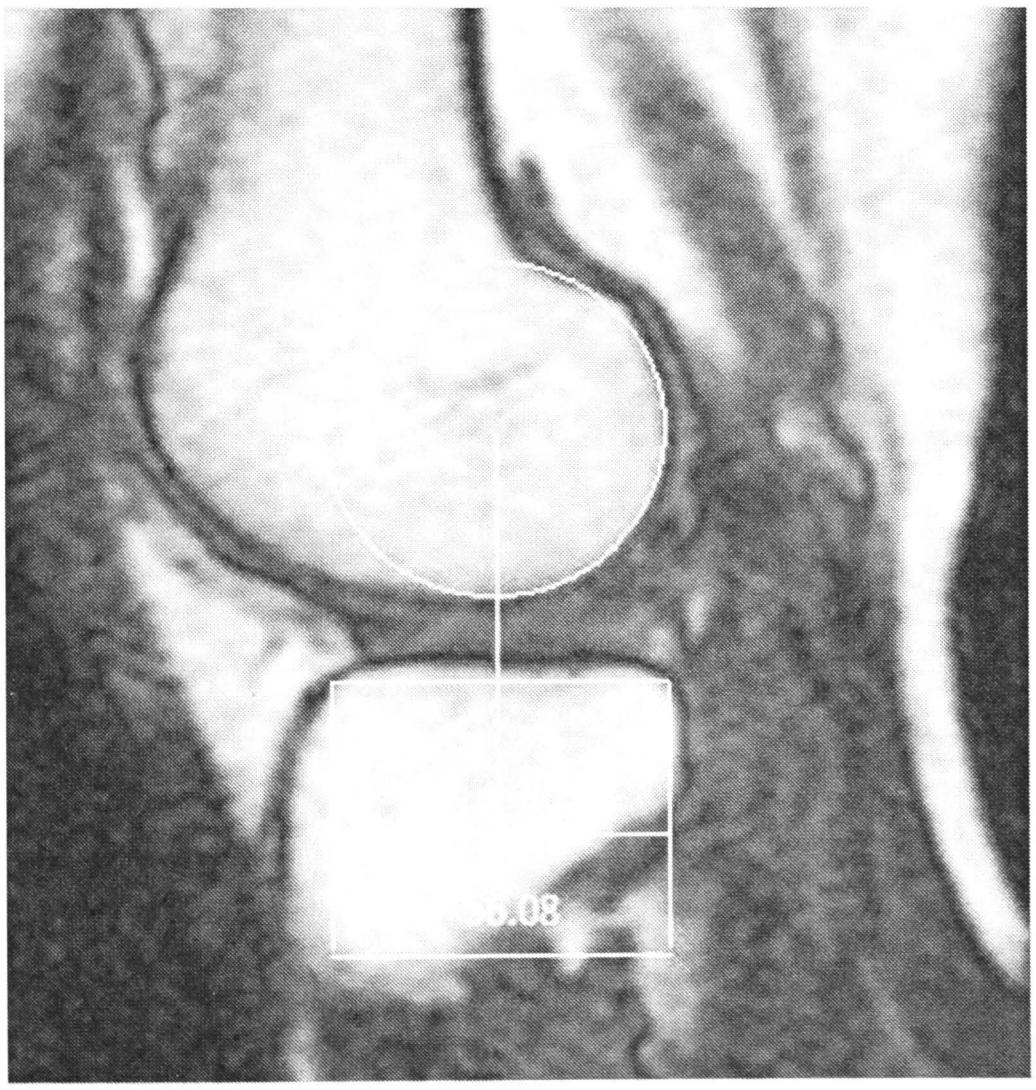


## Flexion facet centre measurement

The position of the flexion facet centre (FFC) over the tibial plateau was located by using a three stage measurement technique with a computer assisted design program (TurboCAD Pro V16.1, D Cheke, IMSI Design) (Figure 3-3). First, the FFC was identified by fitting a circle to define the flexion arc of the posterior condyle. This involved using an arc function to identify 3 points on the posterior aspect of the femur which could then be incorporated into a circle of bit fit. Second, the tibial plateau was defined by a line from the posterior tibial cortex, parallel to the tibial plateau. Lastly, a line was drawn through the FFC perpendicular to the tibial plateau line to measure the

distance from the posterior tibial cortex to the intersection of the perpendicular line. The sagittal slice with the greatest anteroposterior diameter of the medial and lateral tibial plateau was selected to perform the measurements.

Figure 3-3: The position of the flexion facet centre over the tibial plateau was measured in three steps: the arc and centre (FFC) of the posterior femoral condyle were defined, the tibial plateau was defined, and the distance from the perpendicular through the centre to the posterior tibial cortex was determined.



## Precision

The precision of both methods of measurement was tested by repeating measurement from the original scanned images on two occasions at least 24 hours apart. The

precision of mapping the contact points for the medial and lateral compartments was very high with intra class correlation 0.95 (99% confidence interval was 0.92 to 0.96). The precision of measuring of the FFC was also very high with intra class correlation of 0.93 (95% confidence interval was 0.88 to 0.93). The greatest difference observed between the repeated measurements was 0.7 mm for the mapping the TFC point and 0.9 mm for mapping the FFC.

## Statistical Analysis

Statistical analysis was carried out using statistiXL version 1.8 for Microsoft Excel. A two-way repeated measures analysis of variance with Tukey and Scheffe post hoc tests were used to compare the TFC points and FFC positions between the healthy and the PCL deficient groups. A p value of less than 0.05 was regarded as statistically significant.

## Results

Table 3-1 shows the mean and standard deviations for the TFC points and FFC for the healthy and contralateral acute and chronic PCL injured knees.

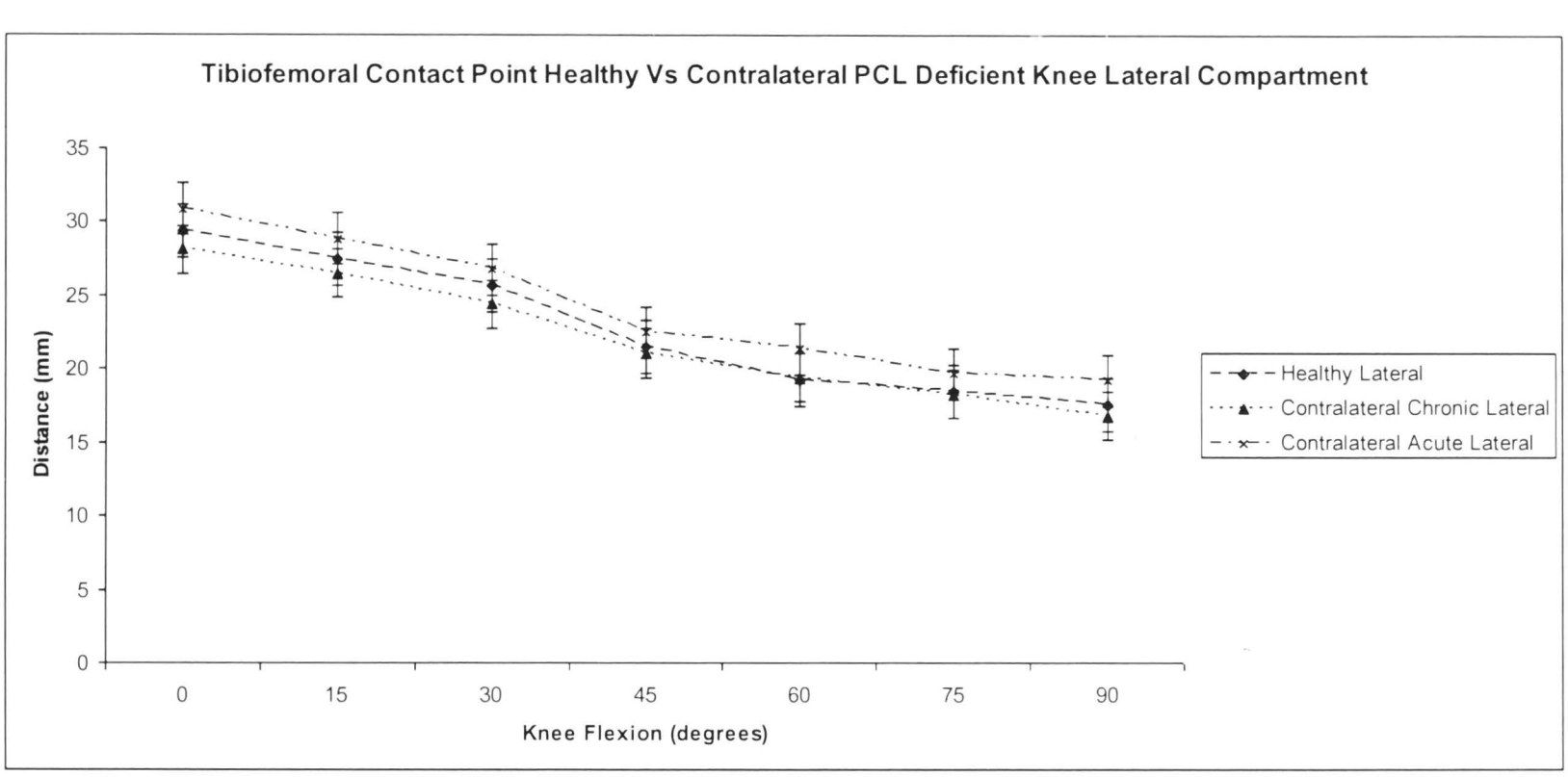
Table 3-1: Mean and standard deviations for the TFC points and FCC for the healthy and contralateral acute and chronic PCL injured knees.

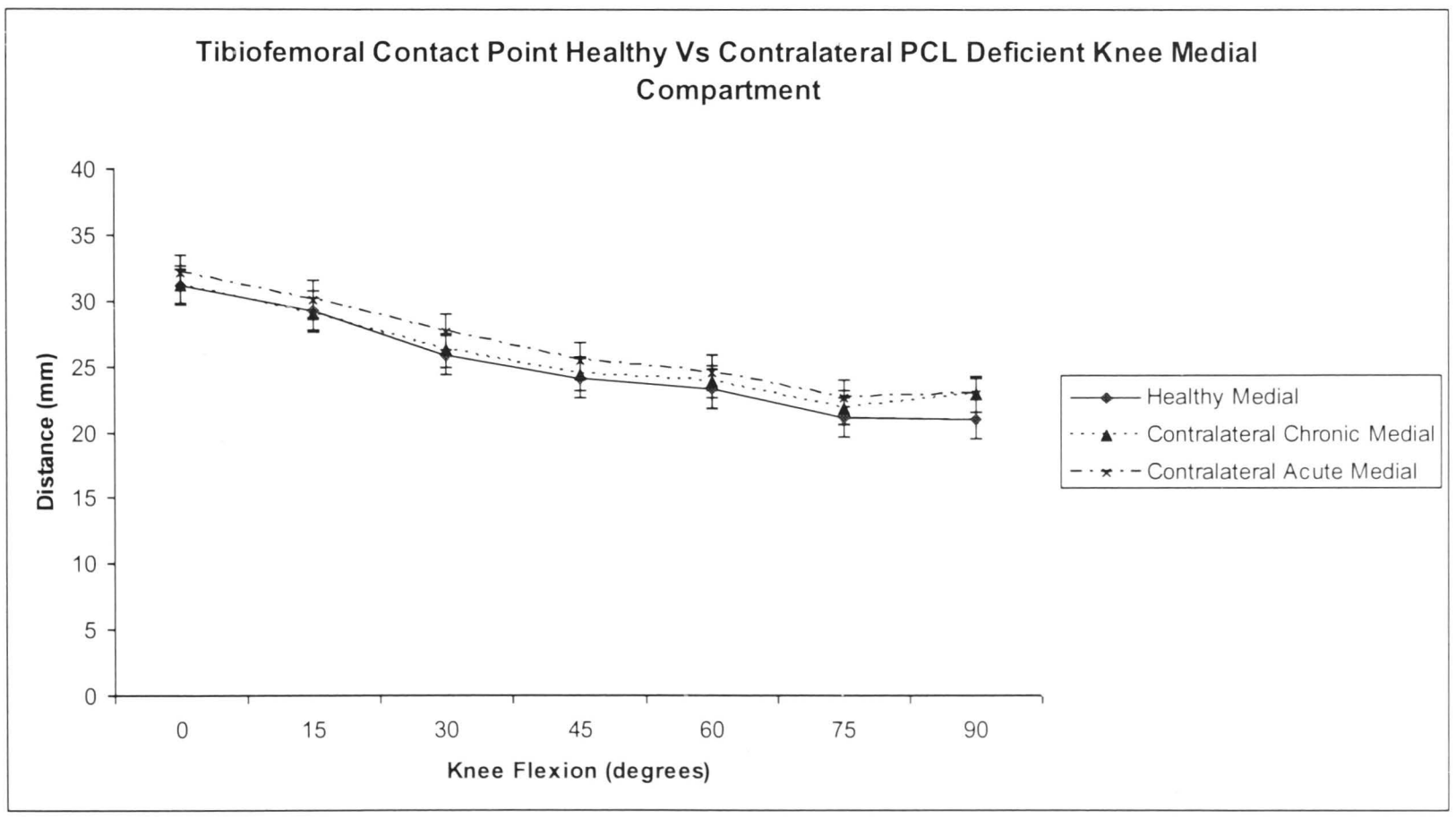
| | Tibiofemoral Contact | | | | | | Flexion Facet Centre | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Healthy | | PCL Acute | | PCL Chronic | | Healthy | | PCL Acute | | PCL Chronic | |
| ateral | Mean | SD | Mean | SD | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| | 29.4 | 2.3 | 28.1 | 1.0 | 30.8 | 1.8 | 25.4 | 1.5 | 25.6 | 2.4 | 27.2 | 1.6 |
| 5 | 27.4 | 1.8 | 26.5 | 0.8 | 28.8 | 1.5 | 23.5 | 1.4 | 23.5 | 2.5 | 25.3 | 1.8 |
| 0 | 25.7 | 1.6 | 24.4 | 0.9 | 26.7 | 2.6 | 22.3 | 1.6 | 22.5 | 2.0 | 24.5 | 1.6 |
| 5 | 21.5 | 1.6 | 21.0 | 1.0 | 22.5 | 1.2 | 20.4 | 1.6 | 20.6 | 1.2 | 22.1 | 1.6 |
| 0 | 19.2 | 1.8 | 19.4 | 1.2 | 21.4 | 1.4 | 17.9 | 1.8 | 19.2 | 1.3 | 19.5 | 0.7 |
| 5 | 18.4 | 1.7 | 18.3 | 1.1 | 19.7 | 1.4 | 17.0 | 1.7 | 17.0 | 1.8 | 18.3 | 1.4 |
| 0 | 17.5 | 1.9 | 16.8 | 1.2 | 19.2 | 1.5 | 16.6 | 1.9 | 15.7 | 1.8 | 17.8 | 0.7 |
| ledial | | | | | | | | | | | | |
| | 31.2 | 1.3 | 31.1 | 1.0 | 32.1 | 1.4 | 20.9 | 1.3 | 20.4 | 0.9 | 22.1 | 1.3 |
| 5 | 29.3 | 1.2 | 29.0 | 0.7 | 30.2 | 1.0 | 22.5 | 1.2 | 21.3 | 0.4 | 22.8 | 0.9 |
| 0 | 25.9 | 1.3 | 26.3 | 0.8 | 27.7 | 1.9 | 22.1 | 1.3 | 21.8 | 0.7 | 23.1 | 0.3 |
| 5 | 24.1 | 1.4 | 24.5 | 0.5 | 25.5 | 1.3 | 21.8 | 1.4 | 21.3 | 0.4 | 22.1 | 0.4 |
| 0 | 23.4 | 1.4 | 23.9 | 1.4 | 24.5 | 1.0 | 20.5 | 1.4 | 20.5 | 0.4 | 22.0 | 1.1 |
| 5 | 21.1 | 1.4 | 21.9 | 1.3 | 22.7 | 1.7 | 20.3 | 1.4 | 19.9 | 1.3 | 21.9 | 1.2 |
| 0 | 21.0 | 1.4 | 22.9 | 1.1 | 22.9 | 2.4 | 20.7 | 1.4 | 19.5 | 0.6 | 21.0 | 0.4 |

Tibiofemoral contact point

In the knees of healthy controls the mean TFC point moved anterior to posterior as the knee flexed from 0 to $90^0$. In full knee extension the medial compartment had a more anterior mean contact point than the lateral compartment. Between 0 and $30^0$ the mean contact point in the medial compartment moved posteriorly by 4.8 mm, which was 0.2 mm per degree. Between 0 and $30^0$ the mean contact point in the lateral compartment moved posteriorly by 3.5 mm, which was 0.1 mm per degree. Between 45 and $90^0$ the mean contact point in the medial compartment did not move posteriorly as much, 3.5 mm in $45^0$, or 0.1 mm per degree. The mean contact point in the lateral compartment also did not move posteriorly as much - 3.4 mm in $45^0$, or 0.1 mm per degree. In the contralateral knee of subjects with an acute PCL injury for any of the mean TFC points in both the medial and lateral compartments from 0 to $90^0$ of knee flexion there was no statistically significant difference when compared to the healthy and contralateral knee of subjects with a chronic PCL injury ($p > 0.05$ at all TFC points). Graphically, the articulation profile of the mean TFC points of all the three groups in the study was similar in both the lateral and medial compartments (Figure 3-4).

Figure 3-4. Graph comparing tibiofemoral contact points in the healthy and contralateral acute and chronic PCL injured knees, performing a leg press against a 150N load through a flexion arc of 0 to $90^0$. The pattern of tibiofemoral contact positions in healthy and contralateral acute and chronic PCL injured knees is not significantly different in both the lateral and medial compartments from 0 to $90^0$ of knee flexion.

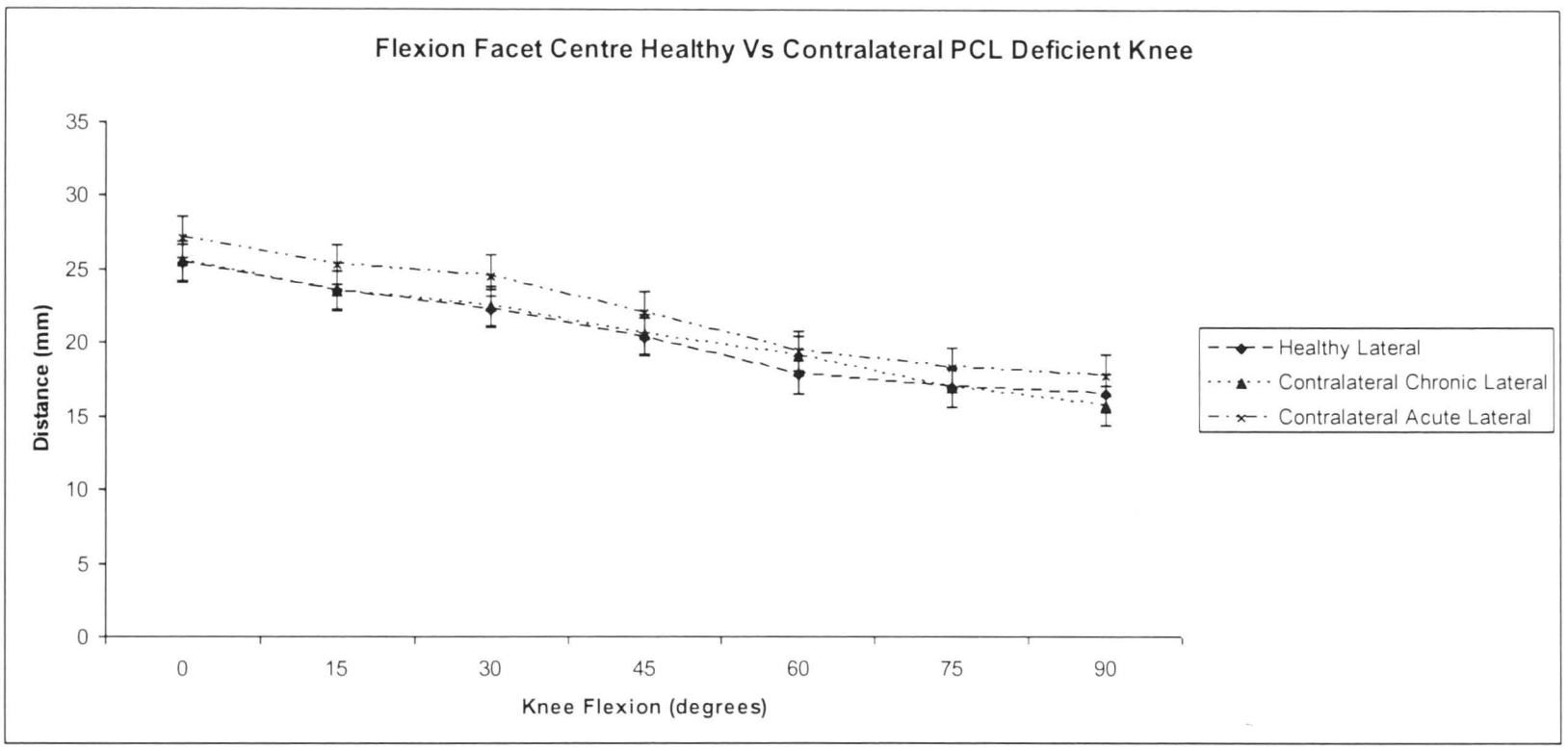


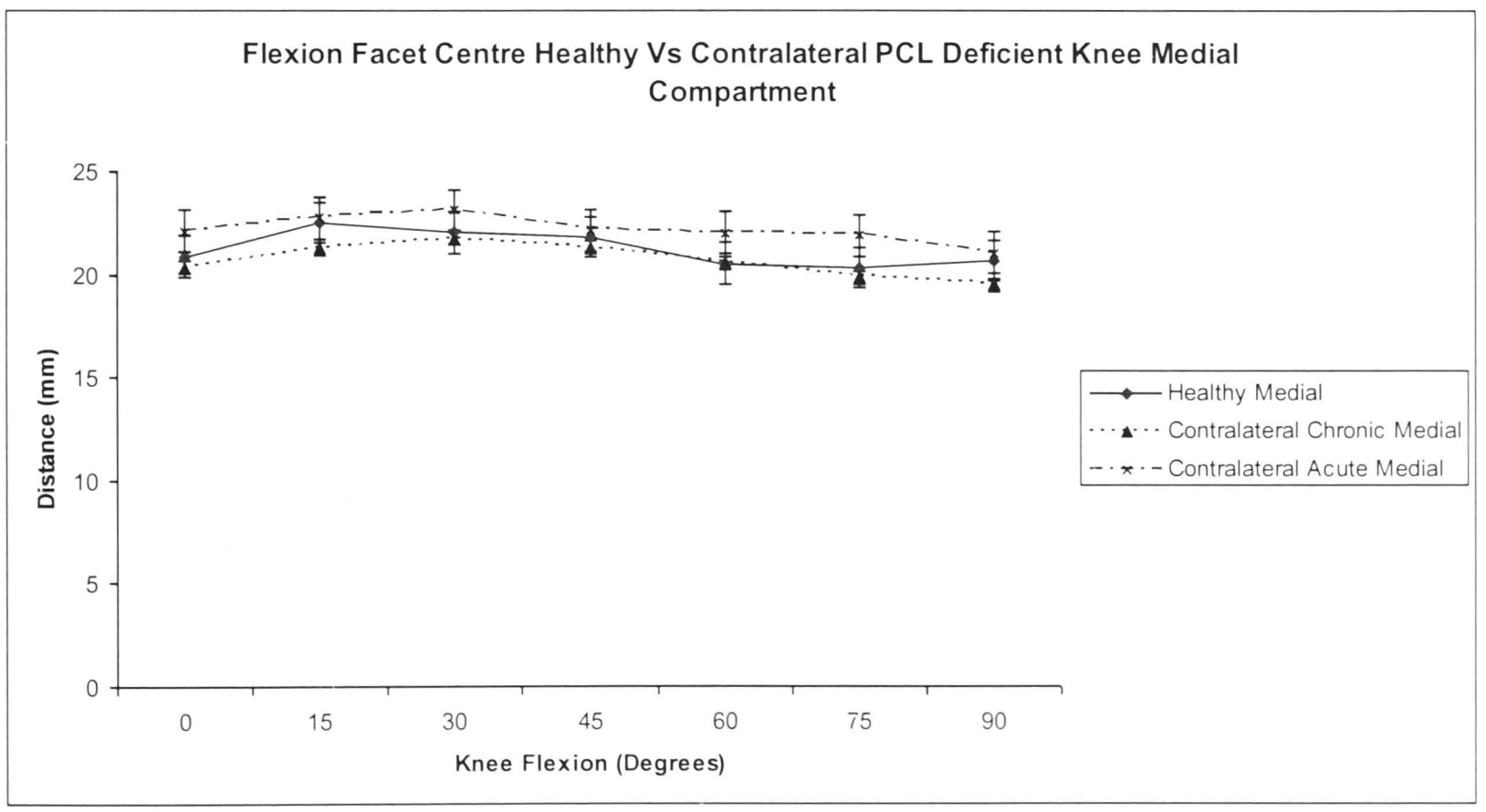



## Flexion facet centre

In the medial compartment the mean FFC was positioned posteriorly over the tibial plateau in knee extension. The medial mean FFC moved anteriorly by 2 mm as the knee flexed from 0 to $15^0$. From 30 to $90^0$ the medial mean FFC returned to its posterior position over the medial tibial plateau. During the entire flexion arc from 0 to $90^0$ the medial mean FFC was positioned over the tibial plateau within a distance of 22.4 mm to 20.5 mm from the posterior tibial cortex. In the lateral compartment the mean FFC moved posteriorly as the knee flexed from 0 to $90^0$. At full extension the mean FFC was located over the tibial plateau 25.3 mm from the posterior tibial cortex. At $90^0$ the mean FFC in the lateral compartment was located over the tibial plateau 16.9 mm form the posterior tibial cortex. The mean FFC in lateral compartment had moved 8.4 mm at an average of 0.1 mm per degree. In the contralateral knee of subjects with an acute PCL injury for any position of the mean FFC in both the medial and lateral compartments from 0 to $90^0$ of knee flexion there was no statistically significant difference when compared to the healthy and contralateral knee of subjects with a chronic PCL injury ($p > 0.05$ at all FFC positions). Graphically, the articulation profile of the mean FFC positions of all the three groups in the study was similar in both the lateral and medial compartments (Figure5-5).

Figure 5-5: Graph comparing flexion facet centre positions in the healthy and contralateral acute and chronic PCL injured knees, performing a leg press against a 150N load through a flexion arc of 0 to $90^0$. The pattern of FFC positions in healthy and contralateral acute and chronic PCL injured knees is not significantly different in both the lateral and medial compartments from 0 to $90^0$ of knee flexion.



## Discussion

The aim of this study was to compare the sagittal plane tibiofemoral articulation of the contralateral knee in subjects with acute and chronic PCL injuries to healthy controls to determine whether it is appropriate to use the contralateral knee as a healthy control and whether the contralateral knee undergoes adaptive articulation due to altered proprioception from the injured knee. This study reported that there was no significant difference in the position of the TFC points and FFCs for both the lateral and medial compartments in the contralateral knee of subjects with acute and chronic PCL injures and healthy controls. The implications of these findings is that the contralateral knee of subjects with PCL deficiency can be used as a valid control in in vivo articulation studies and there appears to be no adaptive changes in the articulation pattern of tibiofemoral joint in subjects with chronic PCL deficiency.

The limitations of this study include supine articulation, small sample size, and the cross sectional nature of the study. The supine leg press was intended to simulate a isometric contraction. It is difficult to extrapolate whether this replicates the forces during sporting or activities of daily living and as such could potentially be a source of error in our results. The reason for the small sample size is that isolated PCL injures are rare and often managed in the community with a minority referred for specialist opinion. This may explain why in vivo studies on PCL deficient articulation generally have small participant numbers[3]. The obvious limitation of the small number of study participants is the reduced power of the study and the difficulty in standardising the groups for age, sex and level of sporting activity. A prospective study that sequentially analysed the articulation of patients with a PCL injury through

the acute and chronic phases would provide a more in depth analysis of temporal changes in knee articulation with a PCL injury. The difficulty with this design however, is the prolonged time period for the study and potential loss of patients to follow up. Nevertheless, the authors plan to follow up the acute PCL patient group over a 2 to 5 year period to better understand the temporal changes in knee articulation with a PCL injury.

There are several studies in the literature that have examined risk factors for ligamentous knee injury[4,5]. The majority of these studies have focused on anterior cruciate ligament (ACL) injuries. These risk factors can be subdivided into anatomical, neuromuscular and familial factors[4]. Anatomic factors include female gender, intercondylar notch stenosis, small ACL volume, increased posterior slope of the tibia and knee hyperextension. Neuromuscular factors include reduced proprioception, reduced quadriceps to hamstring ratio, inadequate muscle stiffness, high degree of dynamic valgus motion in landing and decreased neuromuscular control to the trunk. Although specific familial factors are difficult to isolate the literature suggests that there is at least some genetic component in the risk of sustaining an ACL injury. No studies were found that evaluated whether there was a pre-injurious abnormality in joint articulation pattern in subjects with PCL injuries.

Anatomical and biomechanical studies have shown that the ligamentous structures of the knee not only act as stabilisers but also provide proprioception[5]. In vivo articulation studies have shown that acute PCL injury produces abnormal knee articulation in the medial compartment of the knee. Specifically, there is posterior subluxation of the medial tibia with respect to the femur as the knee flexes from 0 to

$90^{03}$. It is not known whether the contralateral knee modifies its articulation to adapt to altered proprioception from the injured knee. The results from this study show that in subjects in chronic PCL injuries the contralateral knee maintains a similar articulation profile to a knees of healthy controls during an isometric contraction, suggesting there is no adaptation. This is an important finding as abnormal articulation in the medial compartment has been associated with increased chondral and meniscal deformation forces[10].

Several studies in the literature have used the contralateral knee as a control when analysing the in vivo articulation of the PCL deficient knee[3, 11]. However, there have been no studies to determine whether the contralateral knee is appropriate to use as a control. The results form this study demonstrate that the contralateral knee in subjects with both acute and chronic PCL injuries does not have an altered articulation profile compared to a knees of healthy controls and therefore can act as an appropriate control.

## Conclusions

In conclusion, this study found that there was not a significant difference in the articulation profile of the contralateral knee of subject with acute and chronic PCL injuries compared to knees of healthy controls. In the setting of chronic injury the contralateral knee does not modify its articulation profile during an isometric contraction. Importantly, the contralateral knee can be used as a valid control when evaluating the articulation of the PCL deficient knee.

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# Conclusion

The specific aims of this thesis were:

1. To analyse sagittal tibiofemoral joint articulation in terms of tibiofemoral contact (TFC) and the position of the flexion facet centre (FFC) of patients with a PCL injury using magnetic resonance imaging.
2. To analyse sagittal plane tibiofemoral joint articulation in terms of TFC and the position of the FFC of the contralateral knee of subjects with PCL deficiency to determine whether it can act as a valid control or whether an abnormal articulation profile is associated with this injury.

With respect to the first aim, the thesis demonstrated that there is a significant difference in the medial compartment sagittal plane articulation of the tibiofemoral joint in subjects with an isolated PCL injury. Specifically, the contact pattern and FFC was significantly more anterior in the injured knee from 45 to $90^0$ of knee flexion in the medial compartment compared to the knees of healthy controls. The greatest difference between the mean TFC points of both groups occurred at 75 and $90^0$, the difference being 4 mm and 5 mm respectively. The greatest difference between the mean FFC of both groups occurred at $75^0$ of flexion, which was 3 mm. There was no significant difference in the contact pattern and FFC between the injured and knees of healthy controls in the lateral compartment.

With respect to the second aim, the thesis demonstrated that there was no significant difference in the TFC and FFC between the knees of healthy controls and contralateral knee of subjects with acute and chronic PCL injuries in the medial and lateral compartments of the knee. In the setting of acute and chronic injury the contralateral

knee does not modify its articulation profile and the contralateral knee can be used as a valid control when evaluating the articulation of the PCL deficient knee.

The limitations of this thesis include supine analysis and small sample size. The supine leg press was intended to simulate a squat. It is difficult to extrapolate whether this replicates the forces during sporting or activities of daily living and as such could potentially be a limitation to the generalisability of our results. Although the number in the study was small (10 knees of healthy controls and 10 PCL injured knees), the number was sufficient to demonstrate a significant difference in the sagittal plane articulation of the PCL deficient knee. It is however worth noting that other studies have involved similarly small numbers of subjects. With respect to the analysis of the contralateral knee, limitations include small sample size and the difficulty in standardising the groups for age, sex and level of sporting activity. A prospective study that sequentially analysed the articulation of patients with a PCL injury through the acute and chronic phases would provide a more in depth analysis of temporal changes in knee articulation with a PCL injury. The difficulty with this design however, is the prolonged time period for the study and potential loss of patients to follow up.

Future work would include using this MRI model to determine if intervention helps to normalise the sagittal plane articulation profile of patients with a PCL injury. Intervention would include non-operative rehabilitation and operative reconstruction and those with persistent symptomatic instability. Also, as mentioned above, sequentially analysing the articulation profile of patients with a PCL injury would add to the body of information about the natural history of the injury.

The findings of this thesis add to the body of knowledge on the kinematics of the PCL-d knee. This thesis is unique in that it analyses the sagittal plane articulation profile using an MRI model of TFC and FFC. The abnormal articulation pattern may explain the increased incidence of medial compartment degeneration reported in literature in subjects with PCL deficiency. Clinically non-operative and operative interventions that address this abnormal articulation may not only improve instability symptoms but also limit chondral deformation and the predisposition to degenerative disease.

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